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Table of Contents

	Page
The Neurotic Potential, the Neurotic Process, and the Neurotic State—*Lawrence S. Kubie*	1
The Significance of Heart Murmurs in Induction Examinations—*Richard P. Johnson*	13
The Ballistocardiograph in Clinical Medicine—*Harold A. Lyons*	25
A New Pickup for Ballistocardiography—*George A. Sheehan, Jr., Frank K. Priebe, Abraham I. Dranetz, and Glen N. Howatt*	39
Long-acting Heparin Preparation: A Useful Adjunct in Anticoagulant Therapy: A Clinical Trial of Depo-heparin Sodium in 15 cases—*William J. Smiles*	45
A Pressure Method of Investment—*Pierre O. Evans*	53
An Electric Defibrillator for Cardiac Resuscitation—*John H. Stover, Jr*	57
Arterial Blood Culture—*Melvin B. Sullivan, Jr. and Clifford P. Powell*	63
The Dynamics of Lumbar Puncture—*Gordon T. Wannamaker*	67
Myocardial Infarction in Young Adults—*Mason R. Baker and William R. Schillhammer, Jr*	75
Pancytopenia Following the Use of Mesantoin and Thiantoin: Report of a Fatal Case—*Harry A. Weiss and William D. Hawley*	83
Hypogonadism: Puberal Seminiferous Tubule Atrophy—*Richard Lawrence and Robert A. Kallsen*	87
Study of Five Hundred Autopsies in Cases of Tuberculosis—*Merrill C. Davenport and Henry M. Greenleaf*	97
Abdominal Hysteropexy for Uterine Prolapse—*Paul Peterson and Sidney L. Arje*	113
Application of Civil Defense Lessons Learned in World War II—*William L. Wilson*	119
Dentistry in the British Army—*H. J. Higgins and S. H. Woods*	135
The Use of Base Shops for Manufacture and Repair of Medical Appliances—*Bruce R. Heinzen*	141

About the Army Medical Service: Procurement of Professional Officers—
Paul I. Robinson 145
Recently Released Medical Films 147
Public Health Aspects of Biologic Warfare 151
Navy's Color Atlas of Pathology 153
BOOKS RECEIVED 155
BOOK REVIEWS 157

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Richard L. Meiling

Richard L. Meiling, M. D.
Director of Medical Services

UNITED STATES ARMED FORCES
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The Neurotic Potential, the Neurotic Process, and the Neurotic State[1]

LAWRENCE S. KUBIE, *M. D.*[2]

THERE is still a widespread tendency to regard all neurotic difficulties as something curious, strange, and alien. The neurotic process is as inevitable a part of human development as speech and breathing. It arises out of the *neurotic potential*, which is a universal phenomenon, apparently peculiar to man, and highly charged with possibilities for both good and evil. The *neurotic process* which has its roots in this human potential may exist for years in masked forms, but crystallizes ultimately either in episodic *neurotic states* or as a persistent symptomatic neurosis. The understanding of the role of neurotic phenomena in human life began with the clinical study of symptomatic neuroses; yet these dramatic and more easily recognized manifestations of the neurotic process are less prevalent and less culturally significant than is the more universal concealed neurotic process. To each patient his own clinical neurosis is of enormous importance, primarily because it causes him illness and suffering. His neurosis hurts him but his neurotic process hurts those around him. He must save himself from his neurosis. His family, his friends, and sometimes society as a whole, must protect themselves from his neurotic process. Therefore, the masked process is of primary importance to society in general.

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[1] Presented at the Monthly Medical Meeting, Army Medical Center, Washington, D. C., April 1950.

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THE CONCEPT OF NORMALITY AND OF NEUROSIS

Only single acts and moments of life will be discussed here, not the normality or abnormality of a whole personality. Our concepts about individual moments must be clear before personalities as a whole can be discussed profitably. If there is agreement on the simpler problem, there will be some hope of reaching clear agreement about the more complex issue later.

What normality is not.—Alan Gregg has said that the best way to describe something is to begin by explaining what it is *not*. For instance, the concept of normality has nothing to do with statistical prevalence. Because something happens frequently does not mean that it is normal. Cavities in the teeth are not normal merely because 99 percent of the population have them. The common cold is not normal even in a pandemic. The statistician's concept of a "*norm*" has nothing to do with "*normal*" as an index of human well-being. Nor is normality identical with usefulness. A neurotic compulsion to do good is at least as frequent as a neurotic drive toward immoral or destructive behavior. This is fortunate, since otherwise the world would be in an even worse fix than it is. Indeed some of the most useful activities in the world have been performed under the urgent pressure of neurotic drives. Nor has normality anything to do with a sense of comfort. A person can have a severe height phobia; but if he lives on a flat plain where there are no hills or tall trees or high buildings, he will not be aware of this, and he will feel entirely comfortable. Many a kleptomania can mask itself comfortably in a wealthy woman's shopping compulsion. I have seen a severely perverted sexual compulsion diverted both comfortably and usefully to the practice of gynecology. Nor is normality identical with lovableness, because many neurotic qualities may enhance a person's appeal, as for instance the neurotic dependence of certain attractive women. Again normality is not identical either with conformity to any particular culture or convention or with rebellion against it. Too many persons conform for neurotic reasons or rebel for neurotic reasons to use either as an index of normality.

Normality is not identical with or measurable by success. Of this, the most dramatic evidence is the frequency with which men react to success by going into severe depressions. One sees this at all levels and in all forms of work and play. We see it in the tennis player who can never let himself win important tournaments from men he can always defeat in practice. We see it in the businessman who goes into a depression when he earns a million dollars; in the writer who commits suicide when his novel becomes a best seller; in the man or woman who reacts to the launching of what should be a happy marriage by deep and destructive gloom. The world did not have to wait

for psychiatry to discover how often success and fame turn to ashes; but psychiatry has given us some understanding of the reasons for this all-too-frequent human tragedy.

Finally, normality is not clarified by the legal effort to define something called "moral responsibility," nor by the law's emphasis on the importance of knowing the meaning of what we do. Psychotics may have a heightened sense of moral responsibility, and may understand as well as clergymen the moral implications of what they do.

What neurotic does not mean.—To be neurotic is not identical with being queer or eccentric or unusual or rare or illegal or unconventional or rebellious or submissive or foolish or weak or useless.

The essential differences between normal and neurotic.—The definition offered here will be in terms solely of the unstable, dynamic equilibrium between conscious and unconscious psychologic processes which operate continuously to determine the quality of human conduct. A distinction which is based on this is the only one which takes cognizance of a fact which is well known to us clinically, namely, that there is literally no single thing which a human being can think or feel or do which may not be either normal or neurotic, and more often a mixture of the two; and that the degree to which it is one or the other will depend *not on the nature of the act, but on the nature of the psychological forces which produce the act.* This is true of work and play, of selfishness and generosity, of cleanliness or dirtiness, of courage and fear, of a sense of guilt or a sense of virtue, of activity or indolence, of extravagance or penuriousness, of ambition or indifference, of ruthlessness or gentleness, of conformity or rebellion, of playing poker or writing poetry, and even of all supposedly simple instinctual acts. Determining each of these is a continuous interplay of conscious and unconscious psychologic forces; and it is the balance between the conscious and unconscious components of this flux which determines the degree of normality or the degree of neuroticism of any act or feeling or trait.

The following facts are basic: (1) In every moment of human life our conduct, our behavior, our thoughts and our feelings, our decisions and plans, our hopes and purposes, and our reactions to one another are determined by a complex group of psychologic processes. (2) Of some of these psychologic processes we are fully conscious; whereas of others we are wholly unconscious, *and cannot become conscious of them* without the aid of special methods of investigation and evaluation, of which psychoanalysis is the pioneer and still the most important. (3) This basic fact, namely that man operates psychologically on at least two levels, is of more than academic interest. It has fundamental and practical importance in human affairs because the consciously and unconsciously organized levels of the per-

sonality have different characteristics, and exert quite opposite influences on behavior, to wit:

That conduct which is determined predominantly by conscious processes is flexible and realistic. Because its motivations are conscious, they can be influenced by conscious appeals to reason and feeling, by arguments and exhortation, by success and failure, by rewards and punishments. In short, there is present the capacity to learn from experience. Therefore, normal behavior is in the truest sense of the word free, i. e., free to learn and to grow in wisdom and understanding.

In contrast to this, that behavior which is determined by a preponderance of unconscious processes is rigid and inflexible. It never learns from experience. It cannot be altered by argument or reason or persuasion or exhortation or rewards or punishment, and not even by its own successes and failures. Because of its very nature it can never reach its unacknowledged and unrecognized goals; it is insatiable and endlessly repetitive, repeating its errors as often as and perhaps even more often than its successes, and marching rigidly ahead on blind and stereotyped paths. This happens whether the pattern of behavior has brought success or failure, and whether it has been a source of happiness or of unhappiness either to itself or to others. Thus neurotic behavior is a state which precludes learning. It cannot change or develop or grow. It is enslaved.

It would be a mistake to assume from this that any act or thought or feeling is determined exclusively by conscious or exclusively by unconscious forces. Instead a mixture is always at work; and our picture of the neurotic process derives from this fact. Whenever the predominant psychologic forces are conscious, the resulting conduct will merit being called *normal*, because conduct so determined is free to be affected by experience and the person is capable of adapting flexibly to changing external realities. On the other hand, when unconscious forces dominate, or when conscious and unconscious forces pursue incompatible goals, then the behavior which results will deserve to be called *neurotic*, precisely because it will be a rigid, repetitive, ineffectual compromise, serving the needs of neither conscious nor unconscious aspirations and motivations.

If these statements are valid, then we may state categorically that if there were no such thing as unconscious psychologic processes there would be no neuroses. There would not be those outspoken clinical neuroses which manifest themselves in obvious symptoms with which we deal in daily practice as the symptomatic psychoneuroses. Nor would there be those masked neuroses which express themselves insidiously in distortions and exaggerations of the customary patterns of living, nor in those quirks which are looked on as the eccentrici-

ties of normal people, nor in those neurotic processes which result in delinquency.

The unconscious.—When we speak of unconscious psychologic processes, we may mean psychologic processes of which we are unaware but which are readily accessible to conscious self-inspection, or we may mean processes of which it is impossible to become aware without the help of special instruments. The two categories of unconscious activity develop differently and have different significances. Those of which we *can* become aware have dropped out of the field of consciousness simply through repetition. Thus all of the simplest activities of life, such as breathing, sucking, excreting, moving, and crying, were originally random and often explosive acts. Early in life their purposeful execution is learned through repetition by which they become economically organized into synergistic goal-directed patterns. As any such act is fully learned, it can be initiated simply by the contemplation of the goal; and as this happens we gradually become unaware of the intermediate steps which make up the act. This great economy is achieved in the process of learning by repetition. It is in this way that we become able to walk without pondering each step, to talk without working out the movements by which we enunciate each word. It is in this way that the violinist and the juggler and the athlete learn complex chains of synergic movements. It is in this way that our thinking processes acquire seven league boots, i. e., the ability to leap over many intervening steps as we perform complex arithmetical processes. This is the source of intuitive thinking whether in science or the arts. In each case the intermediate steps drop into the background and disappear from consciousness. Yet they remain accessible to conscious self-examination. They are what William James would have called the "fringe of consciousness," or what Freud called the preconscious or the descriptive *sub*conscious, as contrasted with the *dynamic unconscious.*

The dynamic unconscious, however, is no mere limbo of shadows; it is an area of hidden force or rather of whole constellations of forces in psychic life. Such unconscious processes are constantly at work in our lives; yet we cannot become aware of them by ordinary methods of self-observation, because they are hidden from us by vigorous opposing forces within ourselves. Throughout life these processes exercise a powerful influence on human behavior; and it is out of their influence that everything that is neurotic in human affairs has its origin. In this sense everything that we say and do and think and feel serves multiple functions and represents symbolically both the conscious and unconscious levels of psychologic organization. From this we may go further and conclude that if the psychologic conflicts of infancy and childhood could take place in the full light of con-

sciousness, then the neurotic process could never be launched in human life.

Why we "repress" certain psychologic processes in such a way as to render them unconscious is a question which will not be discussed here beyond pointing out that it happens whenever conscious or unconscious feelings of guilt and fear make it impossible for us to discharge internal tensions. When this situation arises we automatically render unconscious the tensions born of conflict, and then express them in disguised symbols, in the symptoms of the *neurosis*.

THE SYMBOLIC PROCESS AND THE NEUROTIC POTENTIAL

Thus the symbolic process which I have in mind includes far more than the symbolism of dreams, which is only a special instance. The human being is capable of two related but different types of symbolic process. One gives him the ability to derive abstract concepts from his experiences, to represent these abstractions in symbols, and thus to express and communicate his purposes, needs, thoughts, and feelings through gestures, sounds, words, and their written symbols. The other symbolic process is the one by which man expresses in disguised forms those psychologic tensions which he is unable either to discharge or to face. The first is the symbolic process of self-expression and of communication through language; the second is an unconscious effort to set misgivings at rest through the symbolic process of self-deception. In the developing infant and child these two symbolic processes have a common origin; and the ability to represent internal experiences through various forms of symbolic activity is the *sine qua non* equally of the neurotic process and of speech. It is not clear whether among lower animals either symbolic process is possible to a significant degree. This is why it is doubtful whether the so-called experimental neurosis in animals, which actually is an emotional disturbance that may occur in human neuroses as well, is identical with the neurotic process itself.

Between the two forms of symbolic representation there is a difference about which we can be quite specific. The difference between the representational process in communication and the representational process in the neurosis is primarily the difference between using a symbol for an internal experience of which we are or can be aware, and using a symbol to express an internal experience of which we are unable to become conscious. The capacity to create and use symbols is identical in both and is essential in both. The difference resides solely in the fact that the relationship of the symbol to the underlying psychologic process is conscious in language, and unconscious in the neurosis. Consequently, the roles of the symbolic representative of those two types of "unconscious" experience differ. In speech the

symbol is like the salesman who represents a firm that is doing a legitimate business. This salesman saves the heads of the firm much time and energy because they do not have to visit every customer themselves, yet these principals are known to the customers and can always be reached by them. On the other hand there is another kind of "salesman," i. e., the representative of a gang of criminals, or the secret agent of a foreign country. Even if he is captured, and even if it is known that he is the agent of criminals, he will not divulge their identity or whereabouts except under pressure, if at all. In the neurosis the relationship of the symbol to the inaccessible unconscious processes which it represents is of this nature. Therefore, we must repeat that if human beings were not able in the first place to abstract their psychologic processes and in the second place to represent these abstractions symbolically, and if in the third place they were not able to render certain unacceptable psychologic processes inaccessible to conscious introspection, there could be no such thing as a neurosis. Together then these three human capacities constitute the neurotic potential.

Thus this human vulnerability to the neurosis, i. e., the neurotic potential, arises out of our capacity for symbolic psychologic function, without which there could be neither a neurosis nor a thinking process, but merely dreamlike sensory imagery, passive echoes of previous perceptions. Like the neurosis, planful action and speech require symbolic processes by means of which sensory imagery can be taken apart and reassembled in new combinations.

THE NEUROTIC PROCESS

Out of this matrix the *neurotic process* emerges gradually and progressively. For each child, it starts the first time when some psychologic experience becomes too painful to think about. Hence it becomes repressed to such an extent that all that is accessible to conscious introspection and all that shows to the world is some combination of thought and behavior and feeling which stands as a symbol for what is buried. This representative or symbol will be simple at first, but with the passage of time and the gradual accretion of new buried problems which are more or less related to the first one, the initial symbol can come to represent many hidden psychologic states; and in turn there can be representatives of representatives, symbols of symbols of symbols, so that as the neurotic process evolves the ultimate linked chain of unconscious symbolic representatives can become very complex.

THE NEUROTIC STATE

The crystallization of the neurotic state out of the neurotic process may be illustrated with a few cases. A courageous, artistic, and

musical woman in her late fifties had been brought up in a cultured home. Through her attachment to her father, a man of learning, she had developed a spontaneous interest in literature and the arts. During her early years these preoccupied her almost to the exclusion of social life, but in later adolescence she married an older man of similar tastes and interests who had been one of her father's outstanding students. It was a good marriage and she gave herself to it wholeheartedly and happily. There were no portents of difficulty, except for a few "harmless eccentricities" of taste and dress. The years went by, however, and in the course of time her husband died, one son was killed in the war, and two of her children had to live on the other side of the world. All of this she weathered, but when her youngest daughter made a happy and suitable marriage, the woman broke down and had to seek help. Retrospectively it then became clear that her devotion to literature and the arts and even to her family had served two groups of inner purposes: one healthy and the other neurotic. From her early years, she had suffered from a secret fear of social challenges. In early childhood she would always vomit before going to a children's party. Without realizing it, her studies, her marriage, her home, her children, and her intellectual and artistic interests, and the eccentricities referred to above had served to mask her phobia almost completely. Consequently, during the long happy years of her marriage, she had been wholly unaware of her lurking neurosis, and it was not until the defense provided by home and family was removed that she again found herself confronted by the unresolved neurotic terrors of her childhood. When this happened, the severity of her anxiety in all social situations forced her to retreat into an unwanted isolation. Her loneliness now depressed her so that she lost her ability to enjoy even the inanimate beauty of music, paintings, or a sunset. Gradually she developed various psychosomatic disturbances, an intractable insomnia, and finally a profound depression. Eighteen years of happy marriage had served her family and community well, but had served the patient badly indeed by masking the highly charged neurotic process which was the hidden legacy of an untreated and unresolved childhood neurosis.

Another woman had grown up with an intense and hostile rivalry with her older brother, of which, however, she was totally unconscious. Because the original hostility was unconscious, she failed equally to realize that it had spread to include all men, so that every relationship with a man was poisoned by unconscious hatred and burdened with a confused inner conflict. In her early adult years this rivalry had masked itself tolerably in the life of a socially active, bachelor-girl, with talented writing and a vigorous participation in liberal politics and other community affairs. Ultimately, however,

this same hostile rivalry with men led her to marry a gifted but weak man, who turned out to be impotent. Again because she had not realized the antecedent steps, she did not realize that she had been drawn to him by the weakness which was a part of his impotence, and which both frustrated her and intensified her secret feelings that to be a woman is to be unlovable. After 2 years of this, her seemingly stable adjustment broke down. Her previous activities could no longer serve their original unconscious purposes. She shut out her many friends, turned away from all community activities, became completely blocked in her writing, and lapsed into a severe neurotic depression.

These examples should make it clear that in spite of a virtual absence of any of the conventional signs and symptoms of a clinical neurosis, an unconscious process may be at work below the surface, the neurotic nature of which proves itself as soon as appropriate circumstances light up the patient's deep problems.

As a last example of how the clinical neurosis, i. e., the neurotic state, can crystallize out of the masked neurotic process which I have tried to describe, I shall trace the relevant fragments in the life of a little boy, from his early years to the precipitation of a frank neurotic episode at the age of 11 years. The child was brought up by intelligent parents who gave him a great deal of thought and attention. Because of the mother's prolonged illness, however, he was left alone a great deal during his first 2 years and had less than the optimal amount of stimulating contacts with human beings. What contacts there were were good, but they were not consistent. As a result he developed as a sober, slow-moving, cautious infant and toddler. This quality colored his neuromuscular and emotional responses for many subsequent years.

As time went on first a brother and then a younger sister were born, spaced at about 2-year intervals. In each case the parents made active efforts to help the boy to accept his natural jealousies and to compensate him for whatever he might feel that he was losing through the birth of these young rivals. His responses to the birth of his brother were excellent. The birth of the sister, however, initiated a more difficult phase in his development. It occurred at that period in life in which it is usual for the first wave of curiosity about bodily matters to reach a peak, focusing sharply on the bodies of parents. This youngster had made many frank and unashamed efforts to inspect them closely and he had inspected himself and his new infant brother for comparative purposes. He did this as naturally and with as little guilt as if he had been comparing toys or colors or clothes or puppy dogs or anything else in his environment. The birth of his sister, however, brought an unhappy change in this.

During his mother's third pregnancy his curiosity shifted from the outside of the body to the inside, i. e., to what was going on inside his mother's body. Thus on one occasion he said frankly that he wished that she had a window in her stomach so that he could see what was going on inside, and when he heard that the baby had been born, he complained, "But it couldn't be; I wasn't there to see." This shift in interest automatically brought its own frustrations to which was added a chain of events, to which he reacted with feelings in which unconscious forces came to play a preponderant role. The first event occurred on the day of the mother's return from the hospital when she developed a severe infection and had to return to the hospital at once with the new infant. Thus to the boy it seemed that she came home bringing the baby and then disappeared with the baby almost immediately. Thereafter she was sick and delirious for nearly 2 months, during which time she was too ill to see the boy.

This initiated many changes, which had a bearing on his ultimate neurosis; and from them I shall select those which best illustrate my thesis. First the boy developed a strong aversion to touching or being touched or kissed by either parent, but more particularly by his mother whom he rejected completely when she finally recovered sufficiently to be brought home. He said to her: "You are not my mommy. You are just a baby's nurse. Daddy's my mommy" (a role his father had faithfully tried to play during the long and anxious weeks of the mother's serious illness). Soon thereafter, on a few occasions the boy insisted that he must grow up to be a lady himself so that he could marry his father.

He showed hostility to the baby sister chiefly by shutting her out entirely. Indeed he would hardly look at her. "If looks could kill" became a literal danger to him, and looking became a forbidden and a hostile act. Indeed the earlier manifestations of an eager and intelligent curiosity about the world around him became totally inhibited. He developed a transient eye-blinking tic, and became acutely shy both with adults and with other children, hanging his head with uncertainty and diffidence in manner and voice. He became more imaginative and more seclusive with a preference for solitude. Clearly these changes were symbolic manifestations of the unconscious conflicts with which he was struggling. Because his parents failed to realize until much later the roots of his difficulty, they limited their therapeutic efforts to the usual common-sense devices for restoring his relationship to his mother and recapturing his earlier natural ease with others. These attempts had only limited success, and the impact on his personality of these buried early experiences persisted. This was the neurotic process at its inception and at work.

When he was 11, an episode happened which precipitated a minor but more acute episode. During a school holiday it was noticed that his earlier eyeblinking had recurred in an exaggerated form; and in addition he now expressed a great deal of anxiety lest something happen to his eyes. For instance he wanted to know what would happen to the other eye if an Indian should shoot an arrow in one eye. This puzzled his parents. There even was talk about whether or not he needed glasses; but a lucky incident brought to light the immediate precipitant of the symptom. By chance they happened to ask him about a little boy who had been his best friend in school. The youngster replied, "I never see him any more. He is a dirty little boy." Since his parents had never told the child that anything was dirty, this surprised them and they asked him why he said this. He answered that it was because his little friend frequently tried to peek through the windows into the girls' washroom. This clarified the problem. The angry and jealous curiosity which he had repressed for all the years since his mother's last pregnancy, his sister's birth, and his mother's long illness had been reactivated by his friend's natural act. To see others do what we ourselves want to do but will not allow ourselves to do always stirs our own repressed impulses. Therefore he had had to reject his little friend, precisely as he had repressed his own natural curiosity. The pressure of the unconscious conflict was now so great, however, that it had to be expressed in certain specific protective, symbolic thoughts and acts, i. e., the protective eye blinking and the obsessional fears of injury to his eyes.

Let me carry the story a little further. Immediate symptomatic relief was easy to give. The parents talked to the boy in simple terms not about his symptoms but about the universality of curiosity about the body, telling stories about themselves and their own surreptitious peeping when they had been his age, and so forth. That night the little boy dreamed an interesting dream about an airplane with two round bulges in which he could not tell whether he was looking at the front or the back, or at the top or the bottom of the plane. After this his eyeblinking and his obsessional concern over eye injuries disappeared with remarkable speed. Thus the parents had relieved that fragment of the boy's unconscious guilt which had its origin in his conflict over his pubescent erotic impulses. Unfortunately they did not at the time realize or deal with the supercharge of destructive hostility which in earlier years had attached itself to his sexual curiosity and because this hostile component was not brought to light at this time, it left difficulties in his adjustment which had to be dealt with later.

What does this story tell us? We see the neurotic potential as an essential part of this child's fundamental human equipment for life.

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A sequence of successive repressions then occurred in areas of highly charged conflicts; and these repressions shaped his developing personality and blocked and warped his interpersonal relationship and even interfered with his intellectual development. Thus for a long time it even interfered with his use of his eyes in reading and studying. Finally, when something happened to touch a match to the timber pile, the buried problem erupted in the form of a typical acute neurotic episode of childhood. Thus out of the potential came the neurotic process, and out of the neurotic process crystallized the neurotic state, as a series of symptomatic acts and fears which represented symbolically the boy's guilt-laden and fear-laden struggle with erotic and destructive impulses of which he had been unconscious. In a remarkably clear and condensed form, this story seems to me to demonstrate my thesis, namely, that the neurotic potential exists as an inevitable consequence of our essentially human capacity to represent symbolically all psychologic experiences, even those of which we are ourselves unconscious; that the neurotic process is an equally natural evolution out of this neurotic potential under the influence of unconscious guilt and fear; and that the symptomatic or clinically categorized neurotic state is nothing more than an episode which may be transitory or recurrent or persistent, but in which the automatic symbolic language in which our unconscious struggles seek expression are accentuated and highlighted.

The Significance of Heart Murmurs in Induction Examinations[1]

RICHARD P. JOHNSON, *Colonel, MC, U. S. A.*[2]

THE value of auscultation and especially the significance of murmurs has been subject to wide swings of opinion in the past. When Laennec invented the stethoscope in 1819, he started a pendulum of opinion swinging that even today has not become stationary. At the beginning of the century, a murmur was often overrated. Mackenzie,[3] in a reaction to this state of affairs said that the stethoscope had done more harm than good to medical practice. He concerned himself with the functional efficiency of the heart rather than with structural abnormalities. Since about 1926, it is believed that Mackenzie was in error. We now know that structural abnormalities are important and may result in cardiac failure. Our present position is a reaction to Mackenzie, and today we put much stress on the finding of a murmur. We have, however, learned to differentiate a physiologic or insignificant murmur from a pathologic or significant one.[4-13] During the last war, 10 percent of the rejectees had cardio-

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[1] Read before the Armed Forces Medico-Military Symposium of the Fourth Army, 6 April 1950, at Brooke Army Medical Center, Fort Sam Houston, Tex.

[2] Brooke Army Hospital, San Antonio, Tex.

[3] MACKENZIE, J.: Heart Disease and Pregnancy. Oxford University Press, New York, N. Y., 1921. p. 62.

[4] WHITE, P. D.; ADAMS, F. D.; and CRAIB, D.: Note on cardiac murmurs. Recommendation for revised terminology. Am. J. M. Sc. 203: 52–54, Jan. 1942.

[5] WHITE, P. D.: Clinical significance of apical heart murmurs. Am. J. M. Sc. 174: 731–738, Dec. 1927.

[6] SHAPIRO, M. J.: Follow-up study of systolic murmurs. Am. Heart J. 17: 416–422, Apr. 1939.

[7] BAKER, L. A.; SPRAGUE, H. B.; and WHITE, P. D.: Clinical significance of loud aortic and apical systolic heart murmurs without diastolic murmurs. Am. J. M. Sc. 206: 31–43, July 1943.

[8] SODEMAN, W. A.: Systolic murmur. Am. J. M. Sc. 208: 106–118, July 1944.

[9] GIBSON, S.: Clinical significance of heart murmurs in children. M. Clin. North America 30: 35–44, Jan. 1946.

[10] MASTER, A. M.: Apical systolic murmur. Arch. Int. Med. 81: 518–533, Apr. 1948.

[11] MASTER, A. M.: Apical systolic murmurs in incipient rheumatic heart disease. Bull. New York Acad. Med. 22: 535–544, Oct. 1946.

[12] LYON, D. M.: Significance of systolic murmurs. (Honyman Gillespie lecture) Edinburgh M. J. 48: 589–618, Sept. 1941.

[13] STERN, N. S.: Systolic murmur. Memphis M. J. 21: 82–86, June 1946.

vascular disease. Of these, 60 percent had rheumatic heart disease and 21 percent had hypertension.[14 15] The most important aids in cardiovascular diagnosis were auscultation and blood pressure determinations.

The question often arises whether a sound at the apex is a systolic murmur, or only a muffled or prolonged first heart sound. Heart sounds do have variable durations.[16 17] When listening to systole and trying to detect a murmur, it must be decided when the first sound ends and whether there is a further sound following it. The important point is that this extra sound has a definite duration, occupying, for example, at least one-fourth of systole, before we can be sure it is a murmur and not a part of the preceding first heart sound.[18]

Having found a murmur it must be analyzed. *First*, is it systolic or diastolic, where is it of maximum intensity and in what direction is it transmitted? This is of the greatest value in localizing its origin. *Second*, what is its intensity? Murmurs can be classified on the basis of intensity into grades from I to VI.[19 20] Grade I is very slight; Grade II is slight; Grade III is moderate; Grade IV is loud; Grade V is very loud; and Grade VI is so loud that it can be heard when the stethoscope is removed a short distance from the chest. The distinction between Grades I and II is that Grade II can be heard immediately, while Grade I can be heard only after listening intently for several beats.[18] The majority of murmurs will be Grade II, III, or IV. This does not imply that the loudness of a murmur is directly related to the severity of a valvular lesion but a loud systolic murmur is usually significant, whereas a slight systolic murmur is usually not significant.[21] Because auscultatory findings are subjective, it is advisable to have any abnormal finding confirmed by at least one other member of the examining team whenever this is practical.

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[14] ROWNTREE, L. G.; MCGILL, K. H.; and FOLK, O. H.: Health of Selective Service registrants. J. A. M. A. 118: 1223–1227, Apr. 4, 1942.

[15] FENN, G. K.; KERR, W. J.; LEVY, R. L.; STROUD, W. D.; and WHITE, P. D.: Re-examination of 4,994 men rejected for general military service because of the diagnosis of cardiovascular defects. Am. Heart J. 27: 435–501, Apr. 1944.

[16] ORIAS, O., and BRAUN-MENÉNDEZ, E.: The Heart Sounds in Normal and Pathological Conditions. Oxford University Press, New York, N. Y., 1939. pp. 66 and 86.

[17] LUISADA, A. A.; MENDOZA, F.; and ALIMURUNG, M. M.: The duration of normal heart sounds. Brit. Heart J. 11: 41–47, Jan. 1949.

[18] LEVINE, S. A., and HARVEY, W. P.: Clinical Auscultation of the Heart. W. B. Saunders Co., Philadelphia, Pa., 1949. p. 145.

[19] FREEMAN, A. R., and LEVINE, S. A.: Clinical significance of systolic murmur; study of 1,000 consecutive "noncardiac" cases. Ann. Int. Med. 6: 1371–1385, May 1933.

[20] LEVINE, S. A.: Systolic murmur; its clinical significance. J. A. M. A. 101: 436–438, Aug. 5, 1933.

[21] WHITE, P. D.: Clinical significance of apical heart murmurs. Am. J. M. Sc. 174: 731–738, Dec. 1927.

Because, with few exceptions, a diastolic murmur means organic heart disease and is, therefore, automatically a cause for rejection, we shall concern ourselves mainly with the more controversial systolic murmurs, except to emphasize a few points concerning the important diastolic murmurs of mitral stenosis and aortic regurgitation.

At the apex a diastolic murmur ordinarily signifies mitral stenosis, but not infrequently the murmur of aortic regurgitation may be heard there. An important differential point is that the diastolic murmur of aortic regurgitation comes immediately after the second heart sound without any pause, while that of mitral stenosis comes

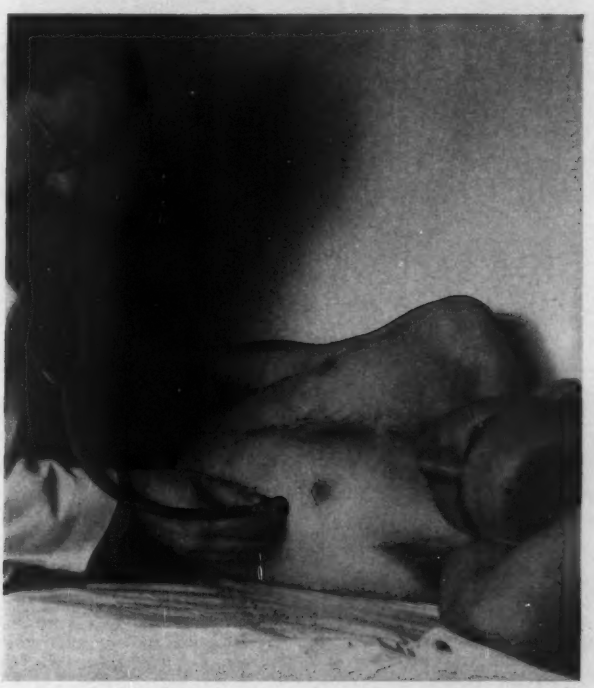

Figure 1.—Physician using the bell chest piece with the patient in the left lateral decubitus position, after an exercise sufficient to accelerate the heart rate to facilitate hearing a faint diastolic mitral murmur.

only after a short interval of silence. No examination of the heart is complete in ruling out mitral stenosis unless the apex has been listened to with the patient in the left lateral decubitus position, after an exercise sufficient to accelerate the heart rate, and using the bell chest piece of the stethoscope (fig. 1). The presence of a forceful first sound at the apex, or a prominent or reduplicated second sound in the pulmonic area, should arouse suspicion of mitral stenosis. Caution

should be observed in diagnosing mitral stenosis solely on the presence of a late diastolic or presystolic murmur.[22 23] It is always better to identify the middiastolic rumble preceding the presystolic murmur before diagnosing definite mitral stenosis.

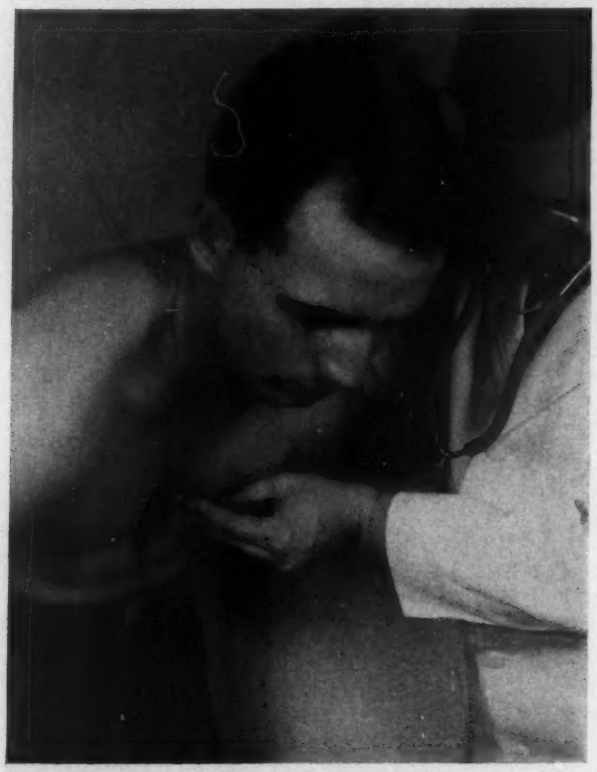

Figure 2.—Physician using the diaphragm chest piece with the patient standing, leaning well forward, and in deep expiration to facilitate hearing a faint diastolic murmur along the left sternal border.

No examination of the heart is complete in ruling out aortic regurgitation unless the patient has been examined either sitting or standing, leaning well forward, in deep expiration, and with the diaphragm or Bowles chest piece (fig. 2). The diastolic murmur of rheumatic aortic regurgitation is best heard along the left sternal border or at

[22] WHITE, P. D.: Heart Disease. 3d edition. The Macmillan Co., New York, N. Y., 1944. p. 81.

[23] ALIMURUNG, M. M.; RAPPAPORT, M. B.; and SPRAGUE, H. B.: Variations in the first apical sound simulating the so-called presystolic murmur of mitral stenosis; a phonocardiographic study. New England J. Med. 241: 631–636, Oct. 27, 1949.

the apex and not in the right second interspace. It is not necessary to demonstrate an enlarged left ventricle, a Corrigan's pulse, a wide pulse pressure, or an abnormal electrocardiogram (EKG) to diagnose aortic regurgitation. It is necessary only to hear its characteristic diastolic murmur occurring immediately after the second heart sound.

Since Mackenzie's day, a common belief has been that a systolic murmur occurring in a heart of normal size, and in a patient without symptoms of myocardial insufficiency, may be disregarded.[24-26] Nothing is farther from the truth when the aim is the detection of heart disease before it has resulted in failure or subacute bacterial endocarditis. Interpretation of the systolic murmur at the apex is usually a difficult problem. It is here that Army experience has taught us the great value of Levine's classification of murmurs, according to intensity. Examinees with a Grade III or louder systolic apical murmur are rejected for military service regardless of the absence of other evidence of heart disease. In the young such a murmur usually signifies rheumatic mitral valve disease, and in the older age groups, left ventricular enlargement.

The difficult problem is the Grade I or II apical systolic murmur. We reject examinees with Grade II murmurs unless the murmur is completely dispelled in certain phases of respiration, and all persons with Grade II murmurs and an authenticated history of rheumatic fever or other sign of heart disease. Grade I or II systolic murmurs may result from fever, anemia, tachycardia, hypertension, hyperthyroidism, nervous excitement, or neurocirculatory asthenia. Another factor occasionally encountered is chest deformity, such as funnel breast or flat thorax. These factors are diagnostically important because if they can be eliminated a Grade I or II murmur may indicate organic heart disease. One important precaution in the evaluation of a Grade I or II apical systolic murmur is to be sure it is not merely a murmur transmitted from the pulmonic area. The site of maximum intensity must be at or near the apex. If an examinee with a Grade I apical systolic murmur has an authenticated history of rheumatic fever we reject him. If there is no history of rheumatic fever or other evidence of heart disease we accept him. In borderline cases in which there is a doubtful or unauthenticated history of rheumatic fever, the extracardiac factors previously mentioned should always be looked for.

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[24] LEWIS, T.: The Soldier's Heart and the Effort Syndrome. Paul B. Hoeber, New York, N. Y., 1918. pp. 66–67.

[25] MACKENZIE, J.: Diseases of the Heart. 3d edition. H. Frowde and Hodder and Stoughton, London, 1918. p. 328.

[26] BOURNE, G.: Cardiac signs in young adults, with special reference to functional murmurs. Lancet 2: 779–781, Nov. 30, 1946.

Life insurance reports illuminate the significance of the apical systolic murmur.[27 28] Considering normal mortality as 100 percent, they show that an inconstant murmur gives a mortality of 111 percent, while a constant murmur gives a mortality of 192 percent. A constant apical systolic murmur loud enough to be transmitted to the left has a mortality of 282 percent. These figures are in hearts normal in size. If there is slight hypertrophy the mortality is 339 percent, and if moderate hypertrophy it is 609 percent. A constant systolic apical murmur plus a history of rheumatic fever gives a mortality of 453 percent in persons without cardiac hypertrophy.

It is advisable to reject examinees with rheumatic heart disease even though their hearts are well compensated and of normal size because (1) with extreme physical or mental exertion they may develop arrhythmia, dilatation and failure; (2) their rheumatic fever may recur; (3) subacute bacterial endocarditis is a constant threat; and (4) they may become a Government charge. It is a serious error to accept a person for military service who has organic heart disease. It is also a serious error to diagnose organic heart disease when it is not present. In the latter case the Government loses a man's service and the man may develop a cardiac neurosis for which cure is difficult.

Physiologic systolic murmurs found at the apex do not indicate organic heart disease and are not a cause for rejection. They can be heard in from 12 to 44 percent of all persons examined.[29-32] If the apex is listened to after exercise, this is increased to over 80 percent.[33] Some of the characteristics of the physiologic systolic apical murmur follow:

1. It is neither loud nor harsh. It is Grade I or II in intensity. It is not loud enough to be heard in the axilla or left base except in a very thin-chested person.[34] Usually, it is heard only over a limited area. Frequently it is maximal medial to the apex, even halfway to the sternum.

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[27] BONNETT, E. C., and LEW, E. A.: Mortality study of systolic heart murmurs. Tr. A. Life Insur. M. Dir. America (1942) 29: 8–75, 1943.

[28] MCCRUDDEN, F. H.: Heart murmurs and insurance. New England J. Med. 204: 598–600, Mar. 19, 1931.

[29] FAHR, G.: Significance of systolic murmur. Nebraska M. J. 15: 458–463, Dec. 1930.

[30] CONTRATTO, A. W.: Significance of systolic cardiac murmurs in college students. New England J. Med. 228: 499–501, Apr. 22, 1943.

[31] O'MEARA, P. J.: Systolic murmurs in healthy hearts based on study of 1,000 naval recruits. J. Roy. Nav. M. Serv. 33: 8–16, Jan. 1947.

[32] MERRILL, J. P.: Auscultatory variations in routine cardiac examination. Mil. Surgeon 102: 487–493, June 1948.

[33] LEVINE, S. A.: Clinical Heart Disease. 3d edition. W. B. Saunders Co., Philadelphia, Pa., 1945. p. 230.

[34] LEVINE, S. A., and LIKOFF, W. B.: Some notes on transmission of heart murmurs. Ann. Int. Med. 21: 298–311, Aug. 1944.

2. It is short, and does not occupy the entire period of systole. Often it begins following a distinct interval after the mitral first sound.[35] [36] It never obscures nor replaces this sound. Usually it terminates before the mitral second sound. This murmur remains short in duration whether the heart rate is slow or rapid, which is not the case with pathologic murmurs which become longer in duration when the heart rate slows.

3. Its intensity is often affected by a change in position, breathing, or heart rate. Usually it is loudest with the patient recumbent, and may disappear when he is upright. Its intensity may vary with certain phases of respiration. It may disappear with inspiration. It may be of decreased intensity with tachycardia, the reverse of murmurs of organic heart disease.

4. Its mechanism is unknown.[37] It is not considered as a diagnosis unless the heart is normal in size and the blood pressure is normal.

An innocent cardiorespiratory murmur is often heard at the apex, in the axilla, or near the angle of the left scapula, and is caused by the heart's action on the adjacent lung causing air either to enter or leave and resulting in a respiratory murmur. By careful attention to the effect of different phases of respiration, this murmur can be diagnosed. It is usually present only during inspiration and is systolic in time. Occasionally it is diastolic and may be heard in expiration.

The pulmonic area has been called the "area of auscultatory romance." Many murmurs occur here,[38] but structural disease of the pulmonary valves is rare. Here a murmur can be disregarded if it is Grade I or II in intensity and if there is no other sign of heart disease. Although all loud murmurs were in their beginning only Grades I and II, murmurs of such intensity are so common in the pulmonic area that they must be disregarded unless the patient can be followed for months or years. With full expiration this physiologic murmur may reach Grade III in intensity, but it often disappears on full inspiration.

The more common important organic cardiac diseases that may cause a pulmonic systolic murmur of Grade III or louder include:

1. *Patent ductus arteriosus.*—Here the systolic murmur is loud and there is, in addition, a diastolic murmur. The two murmurs result in a continuous murmur. Typically there is no interval of quiet.

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[35] EVANS, W.: Mitral systolic murmurs. Brit. M. J. 1: 8–9, Jan. 2, 1943.

[36] EVANS, W. (London): Heart murmurs. Brit. Heart J. 9: 1–15, Jan. 1947; and 9: 225–240, Oct. 1947.

[37] HEBERT, G. T.: Cog-wheel breathing.. Lancet 2: 628–629, Sept. 20, 1930.

[38] CHISHOLM, D. R.: Trigonoidation of semilunar valves and its relationship to certain basal systolic murmurs. Am. Heart J. 13: 362–372, Mar. 1937.

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The systolic murmur is often crescendo to the second sound and the diastolic component is decrescendo from the second sound. This results in a triangulation of the continuous murmur with maximum loudness with the second heart sound.

2. *Interatrial septal defect.*—To diagnose this lesion without the aid of cardiac catheterization, increased pulmonary blood flow must also be demonstrated by roentgenogram or fluoroscopy of the chest. Right bundle branch block in the EKG is of diagnostic value.

3. *Isolated pulmonic stenosis.*—Since cardiac catheterization has become a means of studying cardiac disease, this lesion has been found to be fairly common, especially in the lesser grades.[39-41]

4. *High interventricular septal defect.*—The diagnosis of this lesion without the presence of a thrill is apt to be in error. More commonly this murmur is heard lower down along the left sternal border in the third and fourth interspaces.

We work on a basis that candidates with a persistent systolic pulmonic murmur of Grade III or more should be rejected. The insurance companies report that there is only a 12 percent increase in mortality above general expectation in those with pulmonic systolic murmurs of all grades.[27 28]

In the aortic area the problem is simpler in the young age group. We reject for military service those with a *localized* systolic murmur of any intensity (unless Grade I). It is important to make sure that the murmur is not merely transmitted from the pulmonic area. Its maximum intensity must be in the right second interspace. In the older age groups the problem is more complex and we accept an examinee with a Grade I murmur because the underlying slight dilatation of the aorta is common and is not a cause for rejection. In young adults a soft Grade I murmur in the aortic area may be caused by emotional tachycardia or hypertension resulting from the nervousness and excitement of being examined.[42] This can only be diagnosed by repeating the examination after the examinee has calmed down. This type of murmur is not a cause for rejection.

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[39] GREENE, D. G.; BALDWIN, E. DEF.; BALDWIN, J. S.; HIMMMELSTEIN, A.; ROH, C. E.; and COURNAND, A.: Pure congenital pulmonary stenosis and idiopathic congenital dilatation of the pulmonary artery. Am. J. Med. 6: 24–40, Jan. 1949.

[40] POLLACK, A. A.; TAYLOR, B. E.; ODEL, H. M.; and BURCHELL, H. B.: Symposium on cardiac catheterization; pulmonary stenosis without septal defect. Proc. Staff Meet., Mayo Clin. 23: 516–520, Oct. 27, 1948.

[41] DOW, J. W.; LEVINE, H. D.; ELKIN, M.; HAYNES, F. W.; HELLEMS, H. K.; WHITTENBERGER, J. W.; FERRIS, B. G.; GOODALE, W. T.; HARVEY, W. P.; EPPINGER, E. C.; and DEXTER, L.: Studies of congenital heart disease; uncomplicated pulmonic stenosis. Circulation 1: 267–287, Feb. 1950.

[42] HARRIS, T. N.: Cardiologic criteria for diagnosis of rheumatic heart disease in apparently healthy subject. Ann. Int. Med. 26: 554–560, Apr. 1947.

Auscultation along the left sternal border is especially important in the detection of aortic regurgitation and interventricular septal defect. We reject examinees with a localized systolic murmur of Grade II plus or greater unless the murmur is dispelled by full inspiration.

SUMMARY

Figure 3 shows the grades of intensity of systolic murmurs which we consider to be a cause for rejection. The rings designate the area where the murmur is heard with maximum intensity. The position of the patient and the proper stethoscopic chest piece are important aids in detecting the faint diastolic murmurs of mitral stenosis and aortic regurgitation in patients without enlarged hearts. In evaluating

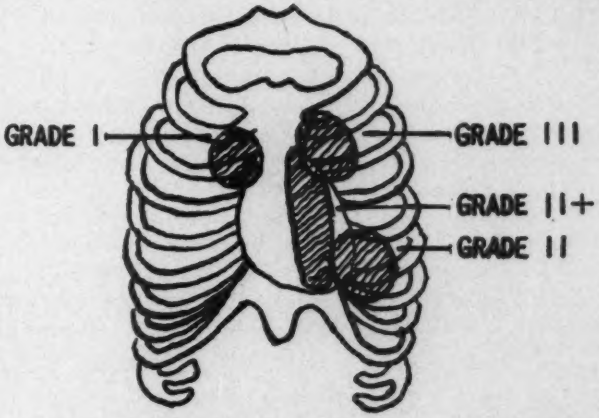

Figure 3.—Diagram showing the area of maximum audibility and the degree of intensity of systolic murmurs that should arouse suspicion of heart disease.

systolic murmurs the accurate localization of maximum intensity, classification of murmurs according to intensity, and the duration of the murmur are important. A satisfactory working rule is to consider as pathologic, until proved otherwise, any of the following systolic murmurs: Grade I aortic, Grade II apical, Grade II plus parasternal, and Grade III pulmonic.

DISCUSSION

JOHN G. KNAUER, *Colonel, MC, U. S. A.*

The sounds and murmurs produced by the human heart lie in and below the lower range of human audibility. It requires, therefore, but very slight impediments in any given case to result in partial, incomplete, or distorted audition of the sounds present. Although investigators have long recognized that the ear is not an ideal modality

for the detection, recognition, and analysis of sounds, it is still possible to improve the power of the auscultation by practice, education, and the use of proper instruments for auscultation. Examinations should be conducted in a quiet room, but it is not always possible to secure a quiet room and, indeed, ofttimes successful diagnoses have been made even in the presence of considerable noise. It is important to differentiate significant heart murmurs but before that can be accomplished it is first necessary to hear the murmurs and to hear them well. Ear pieces for stethoscopes are available in 20 or more sizes. Different shapes can also be obtained. It is thus possible to have a fitting of ear pieces so snug as to eliminate practically all outside noises, and so comfortable as to enable one to use the stethoscope for many consecutive hours without discomfort.

The acoustics of the two common types of chest pieces vary greatly. Many can hear clearly with one kind of bell and not with another. Various types of diaphragm scopes differ even more greatly as to their acoustic properties. If a diaphragm type is used, care should be exercised to see that there is not a bulging, concave, or cracked part. Otherwise many noises will be heard that cannot be attributed to the heart. The rubber or plastic tube of the stethoscope should be substantially thick, new, and elastic; neither too flexible nor too rigid. Stethophones and other amplifying devices are necessary only for examiners who have defective hearing. If such devices are used, it is necessary to exercise even more care than with the ordinary stethoscope to avoid errors because of the introduction of extraneous sounds.

Whatever auscultatory device you may use to assist in examining for heart murmurs, it is important to know that there are many murmurs than cannot be heard at all or at least not clearly unless the examiner moves his head up and down during the examination. A position can thus be found which is the best position for the audition of any given murmur. The best way to miss hearing certain pathologic murmurs is to neglect this simple procedure. In induction examinations there are often many circumstances in which a physiologic third heart sound is confused with a low-pitched diastolic murmur. In such instances, if there should be any doubt as to which of the two is present, the inductee should be examined in a recumbent position with his lower extremities elevated. This maneuver will automatically increase the intensity of a physiologic third heart sound. Muscle twitching in many nervous people tends to interfere with the true heart sounds and may even simulate murmurs. The person being examined should hold his breath in midexpiration and relax his shoulders. This usually relaxes him and the muscle twitching ceases.

If the ribs are prominent and the chest wall thin, the interspaces are often deep or retracted. When this is true, whether you are using a bell or a diaphragm chest piece, the apex beat may slap the chest piece with each systole thus giving rise to vibrations often mistaken for a cardiac murmur, but more frequently mistaken for a friction rub. When the cause is understood and looked for jumping to erroneous conclusions in such instances can be avoided. Split first and second sounds are commonly heard in normal hearts but are frequently mistaken for murmurs. Split sounds are always close together. Split first sounds are commonly misinterpreted as presystolic murmurs. They must be carefully differentiated. The pitch of a split sound is almost invariably much higher than the pitch of a presystolic murmur. The duration is shorter and the quality is totally different—it is more metallic. Split second sounds are more frequent in the presence of heart disease but may still occur in normal people. The presence of a split second sound should prompt us to look for real murmurs. The use of exercise or a whiff of amyl nitrite together with examination in the left lateral recumbent position often facilitates the detection of such murmurs.

The inching technic is useful for the purpose of timing diastolic murmurs. This technic consists simply of rhythmically changing the position of the stethoscope from base to apex or from apex to base. At the base the second heart sound is usually loud, clear, and of a distinctive quality. By noting the rhythm the second sound can thus be positively identified all the way down to the apex. Any murmur following it is a diastolic murmur.

The venous hum, often mistaken for a heart murmur, is rarely heard except in children. It does occur, however, in adults and especially in thin-chested ones. Although this hum is generally heard only in the neck, it is sometimes heard over the entire chest or portions of it. There is a certain quality which, to the initiated examiner, usually distinguishes a venous hum from a cardiac murmur, but if there is any doubt the hum can be eliminated by exercising light pressure at the upper part of the jugular vein. True murmurs cannot be eliminated or modified by this maneuver.

High-pitched murmurs are heard best by the use of a diaphragm type of chest piece. The bell is generally preferred for the low-pitched murmurs. That is why both types of chest pieces should be available. Along with the detection and evaluation of murmurs, it is necessary to consider the characteristics of the first and second heart sounds. If the heart is normal both sounds are proportionately increased after exercise, inhalation of amyl nitrite, or similar maneuvers, and both sounds are proportionately decreased in a recumbent

position. Any change of relative intensity of either heart sound as contrasted with the other is important and may signify the presence of a cardiac lesion. When such a finding is made in the presence of a doubtful murmur, the need for reexamination and study of the inductee is indicated.

The Ballistocardiograph in Clinical Medicine

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WILLIAM HARVEY started an unending series of studies of the circulation in 1628 with his epic, "Concerning the Motion of the Heart and Blood." From these studies various types of apparatus have been devised to penetrate the secrets of the circulation and the heart, e. g., the pulse recorder, electrocardiograph, plethysmograph, cardiograph, stethocardiograph, the cardiac catheter, and the electrokymograph. All these are used to study the heart or the pressure produced by its systole in health and disease. The ballistocardiograph records the motion imparted to the body by blood leaving or entering the heart, but not its pressure. It is an ingenious device first used in 1877 [2] but greatly improved by Starr,[3 4 5] who used a bed suspended by wires from the ceiling, its motion damped by a spring. The movement imparted to the body by the rush of the blood is not detected on physical examination except in patients with aortic regurgitation. When we stand on a spring scale the motion is apparent as a jiggle synchronous with the pulse. The mechanical principle involved is found in Newton's Laws of Motion.[4] Simply stated, to every reaction there is an opposite and equal reaction. When blood leaves the heart or impinges on the walls of vessels it exerts forces which move the body. This has recently been described by Starr.[6]

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[2] GORDON, J. W.: Certain molar movements of human body produced by circulation of blood. J. Anat. Physiol. 11: 533, 1877.

[3] STARR, I.; RAWSON, A. J.; SCHROEDER, H. A.; and JOSEPH, N. R.: Studies on estimation of cardiac output in man, and of abnormalities in cardiac function, from heart's recoil and blood's impacts; ballistocardiogram. Am. J. Physiol. 127: 1–28, Aug. 1939.

[4] STARR, I.: The Ballistocardiograph—An Instrument for Clinical Research and for Routine Clinical Diagnosis. Harvey Lecture Series XLIII, 1946–47. pp. 194–220.

[5] STARR, I.; and RAWSON, A. J.: Vertical ballistocardiograph; experiments on changes in circulation on arising, with further study of ballistic theory. Am. J. Physiol 134: 403–425, Sept. 1941.

[6] STARR, I.; HOROWITZ, O.; MAYOCK, R. L.; and KRUMBHAAR, E. B.: Standardization of ballistocardiogram by stimulation of heart's function at necropsy; with clinical method for estimation of cardiac strength and normal standards for it. Circulation 1: 1073–1096, May 1950.

The apparatus of Starr is the classical ballistocardiograph used in many institutions. Some of the modifications [7-9] of this apparatus with their recording devices have made elaborate installations necessary for the bed and for the recording equipment, and some require an electrophysicist to keep them in operation. Dock and his collaborators [10 11] have described technics for obtaining records directly from the body at rest on any solid bed or table, thus making ballistocardiography available to the practicing physician. I have used these technics and have been associated with Dock in their development. The ballistocardiograph in this form requires no special skill for operation and can be constructed easily by anyone with the interest and desire to have a ballistocardiograph. After procurement of the several parts, the instrument can be constructed in several hours, using a knife, a screwdriver, and a soldering iron. The recordings with such instruments are essentially the same as those made with Starr's apparatus on the same patients.

THE APPARATUS

The first apparatus developed consisted of an ordinary pulse capsule applied to the vertex of the head with a counterweight. Although the recorded waves are of a fair quality, head tremors and kyphotic spines cause bizarre records. A better technic is to take recordings from the shins by the use of a photoelectric cell (figs. 1, 2, 3, and 4), or to record the velocity of the motion of the body by the use of a coil and a magnet, the coil being fixed to the shins and the magnet to the table, so that the movement of the body induces a current in the coil (figs. 3, 4, 5, and 6). The dry cells and the electrical connections are placed inside the balsa blocks of which the apparatus is constructed. In the photoelectric cell type the light beams are interrupted on the photocell by a screen. In the electromagnetic type the magnet is fixed by the stand on the table. The coil is mounted in or on the balsa wood. The electromagnetic and the barium titanate piezoelectric method (another described ballistocardiograph)[12] are the simplest,

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[7] NICKERSON, J. L., and CURTIS, H. J.: Design of ballistocardiograph. Am. J. Physiol. 142: 1–11, Aug. 1944.

[8] NICKERSON, J. L.; WARREN, J. V.; and BRANNON, E. S.: Cardiac output in man; studies with low frequency, critically-damped ballistocardiograph, and method of right atrial catheterization. J. Clin. Investigation 26: 1–10, Jan. 1947.

[9] BROWN, H. R., JR., and PEARSON, R.: New electronic method for simultaneous recording of ballistocardiograph and electrocardiograph. Am. Heart J. 35: 756–762, May 1948.

[10] DOCK, W., and TAUBMAN, F.: Some technics for recording ballistocardiogram directly from body. Am. J. Med. 7: 751–755, Dec. 1949.

[11] DOCK, W.: N. Y. State J. Med. (in press).

[12] SHEEHAN, G., JR.; DRANETZ, I.; PRIEBE, F. W.; and HOWATT, G. N.: Practical clinical application of new piezoelectric material ballistocardiograph. Bull. N. Y. Acad. Med. 26: 263, Apr. 1950.

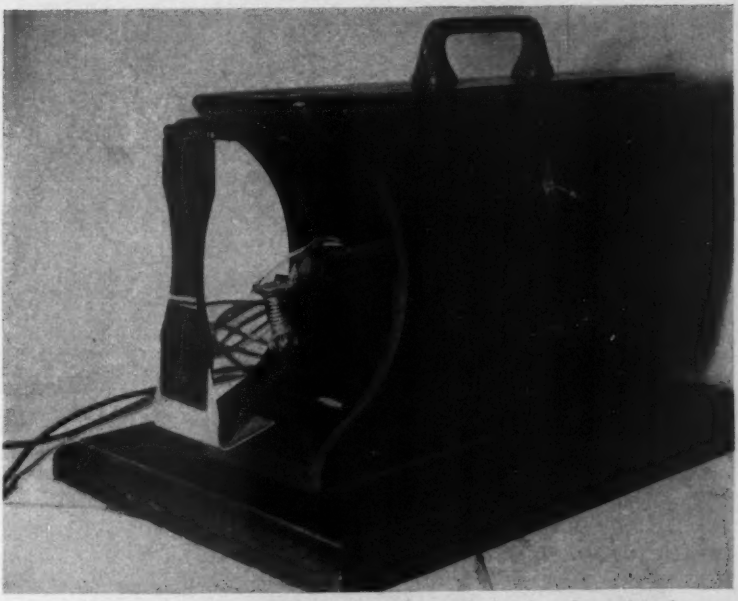

Figure 1.—The photoelectric cell ballistocardiograph showing the various features of the instrument.

Figure 2.—The method in which the photoelectric cell type ballistocardiograph is operated.

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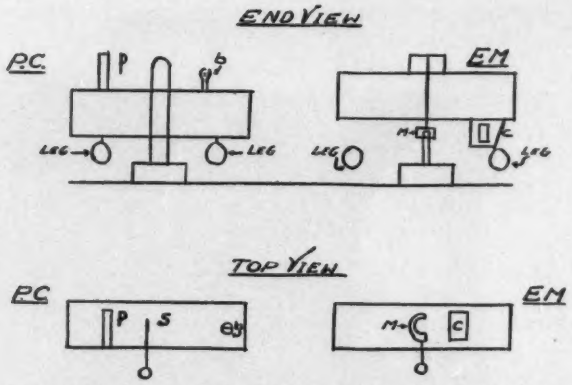

Figure 3.—Diagram of the end view and top view of the photoelectric cell type (PC) *of the apparatus and the electromagnetic type* (EM). (p) *Photocell;* (b) *3-volt* (2-cell) *bulb;* (m) *magnet;* (c) *coil; and* (s) *screen for the photoelectric cell.*

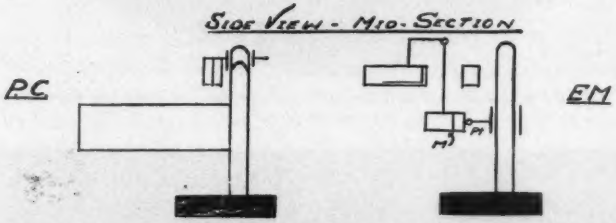

Figure 4.—Side-view diagram of the photoelectric cell type and the electromagnetic type apparatus.

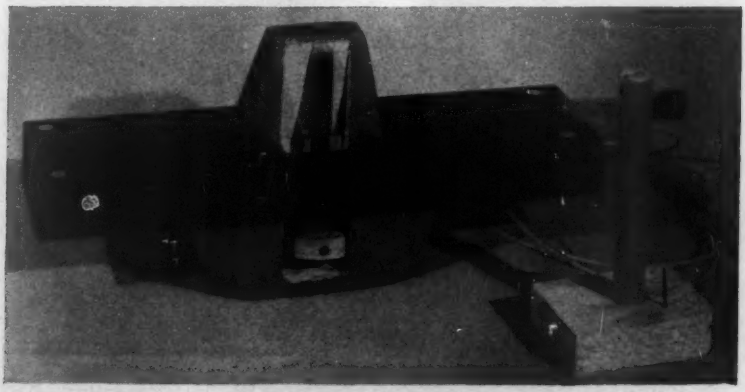

Figure 5.—The electromagnetic type of ballistocardiograph with the stand containing the point for fixing the magnet at the dot on the magnet.

Figure 6.—The electromagnetic type of ballistocardiograph in operation.

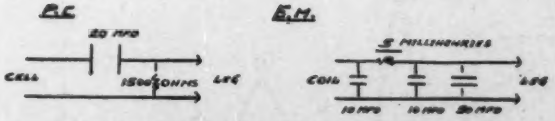

Figure 7.—Wiring diagram of the photoelectric cell and the electromagnetic ballistocardiographs. The 1,500 ohms resistor in the PC type is used only when recording on an amplifying recorder. The microfarad resistors in the EM type are used only when recording on a string type galvanometer.

and are easily adaptable to all types of recording apparatus, whether a string, beam, or direct writing galvanometer, or an oscilloscope. Many other arrangements of the electromagnetic and photoelectric cell have been obvious but the simplest have the elements mounted on 10-inch balsa blocks which are placed across the shins, while a base between the legs either fixes the magnet mounted on a hinge, or casts its shadow on the photoelectric cell (figs. 3, 4, 5, 6, and 7). The photoelectric cell apparatus produces a record purely by displacement whereas the magnet type records the velocity of the body movements. The latter is more informative in degenerative heart disease, the former in coarctation and peripheral shock. The tracings illustrated in this article have all been made with one of the types of ballistocardiographs described in the foregoing. All tracings are accurately reproducible, and records can be identified as belonging to a particular person because each of us has a ballistic tracing as characteristic as our profiles.

THE NORMAL BALLISTOCARDIOGRAM

The footward and headward movements of the body are recorded respectively as downward and upward deflections of the tracing (fig. 8). Using Starr's original designations of the waves, the first wave is an H wave which is considered to be caused partly by auricular systole and by the apex thrust of the heart. The next wave is the downward I wave which is caused by the recoil from ventricular ejection of blood. This is followed by the upward J wave which is caused by the impact of blood flow on the aortic arch and pulmonary bifurcation. The downward K wave which follows is caused by deceleration of the blood in the descending aorta and impact on its bifurcation. This is followed by a series of waves caused by after-vibrations or return flow into the ventricles in diastole. They are

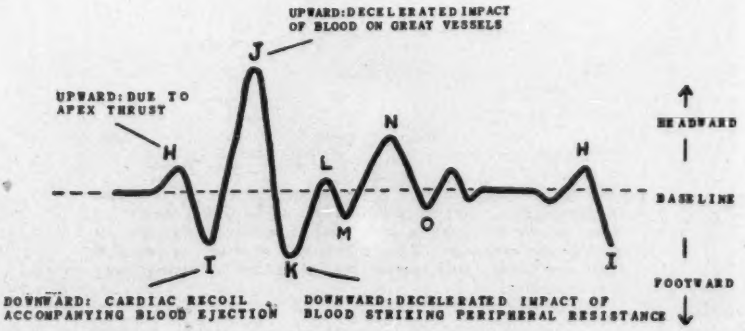

Figure 8.—Drawing of the normal ballistocardiographic wave pattern with a brief explanation of the most significant waves.

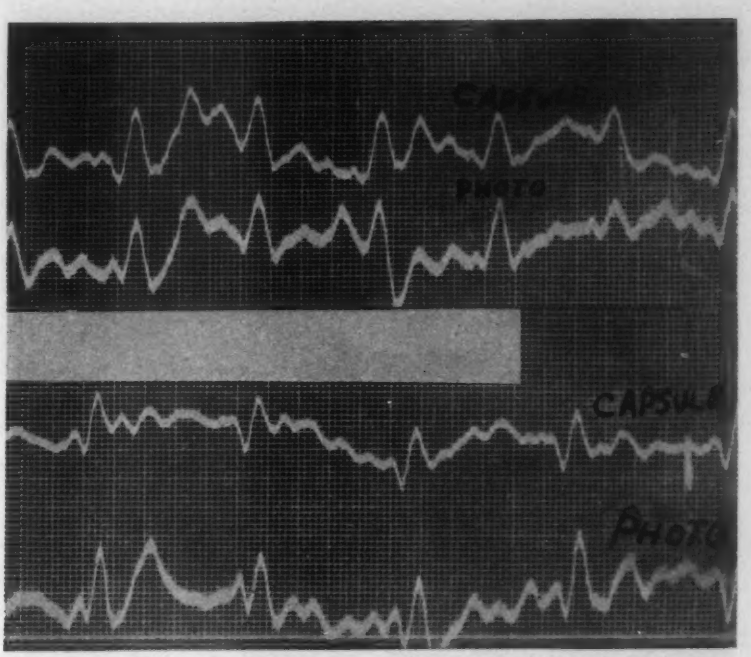

Figure 9.—Normal ballistocardiograms recorded simultaneously using the capsule and the photoelectric cell method.

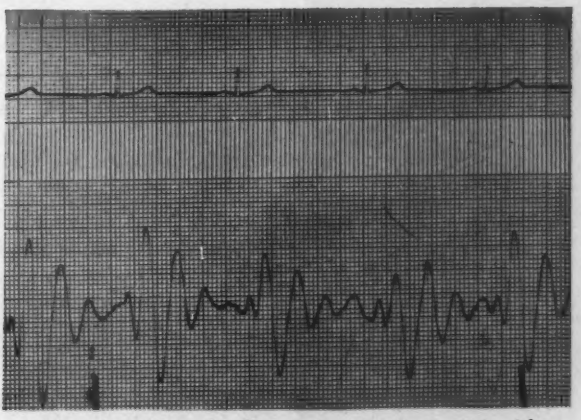

Figure 10.—Normal ballistocardiogram (magnetic) with simultaneous EKG.

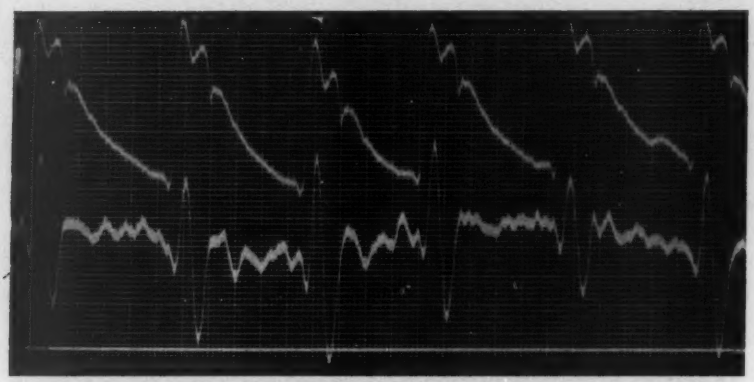

Figure 11.—Normal ballistocardiogram with simultaneous carotid artery pulse (photoelectric).

lettered successively L, M, N, O. Records obtained with this apparatus have been of value in diagnosis, prognosis, and management. There are minor variations in normal ballistocardiograms as there are in normal electrocardiograms, but fundamentally they do not vary greatly. A series of normal ballistocardiograms of different persons are shown (figs. 9, 10, and 11). The various waves are easily identified. A correlation of the various mechanical effects of the cardiac cycle can be recorded simultaneously (fig. 12).

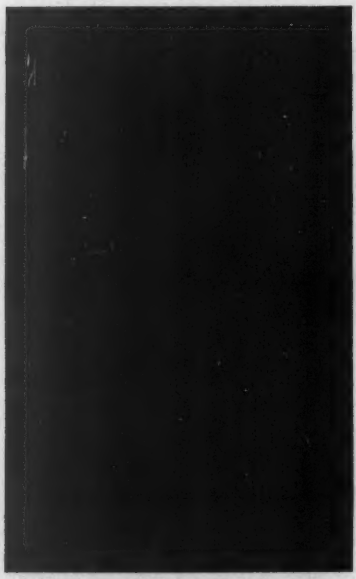

Figure 12.—Simultaneous record of normal electrokymogram, ballistocardiogram, and stethocardiogram (reading from top to bottom).

ABNORMAL FORMS

The abnormal ballistocardiogram reveals itself in either (1) a pronounced variation with the respiratory cycle, or (2) a low or high amplitude, (3) notched J waves, (4) deep or absent K waves, (5) poor definition of the complexes or a mere jumble of oscillations, or (6) a combination of the variations. With some mitral lesions or gallop rhythm, large diastolic waves, exceeding the I J K, may be encountered. Examples are shown in figures 13 and 14. Many patients with angina pectoris or asymptomatic healed infarcts show a typical pattern in

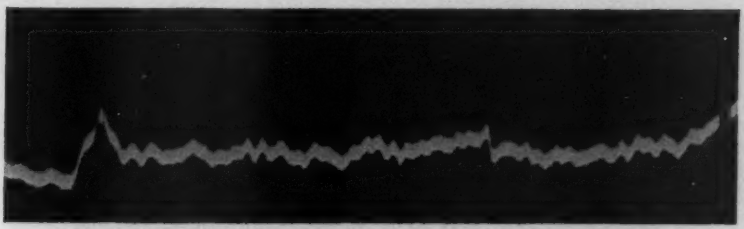

Figure 13.—Ballistocardiogram of a patient with extensive infarction of the anterior myocardial wall (photoelectric).

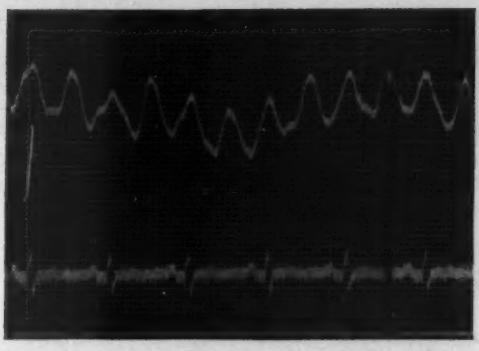

Figure 14.—Ballistocardiogram showing the undulatory type of pattern in a patient with pericardial effusion.

which there is lowered amplitude, poor definition, or both occurring in the expiratory phase of the respiratory cycle. Brown [13] has classified this into groups, depending on the degree of change. He has pointed out the rough correlation between the degree of change and the severity of the disease process. The presence of this pattern is a valuable aid in the diagnosis of coronary disease and may be the only evidence of such when electrocardiograms and exercise tests reveal no abnormalities. The pattern may be improved by abdominal binders and about 25 percent of patients with angina pectoris can be relieved of their symptoms, provided a special abdominal corset converts the abnormal ballistocardiogram to a normal or more normal tracing. A record is made with the patient recumbent and not upright. We have 12 patients who have benefited by the corset with an increase in exercise tolerance and no further need for nitroglycerin

[13] BROWN, H. R., JR.; HOFFMAN, M. J.; and DE LALLA, V., JR.: Ballistocardiographic findings in patients with symptoms of angina pectoris. Circulation 1: 132–140, Jan. 1950.

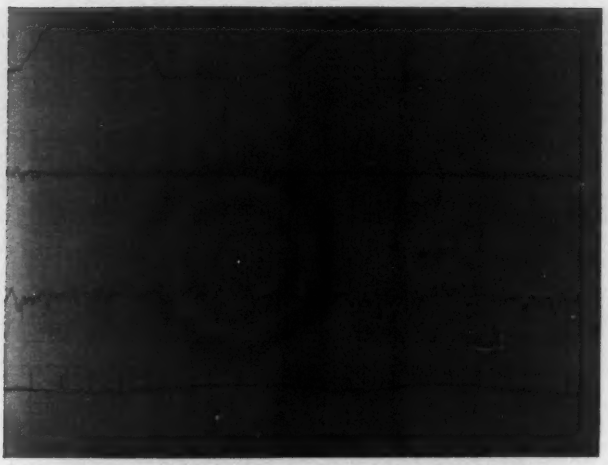

Figure 15.—Ballistocardiogram showing the effect of an abdominal belt in a patient with angina. The respiration is also recorded.

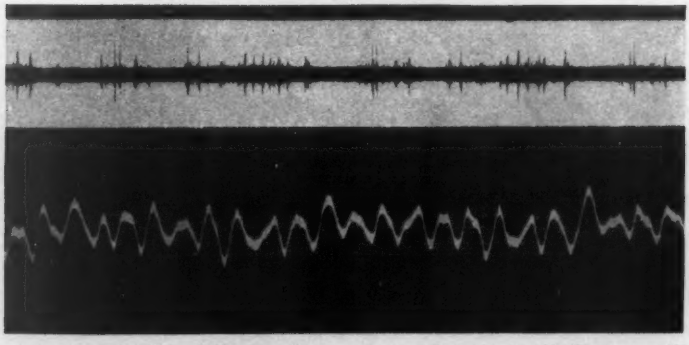

Figure 16.—Ballistocardiogram in a patient with auricular fibrillation, with simultaneously recorded heart sounds. Note the bizarre pattern and greater amplitude associated with louder sound.

(fig. 15). Some patients with intractable angina have been spared sympathectomy by the use of the corset.[14] Brown [15] has evidence obtained by catheterization studies in animals that increased venous return to the heart is the probable explanation for the improvement. Our own limited experience has demonstrated that in humans the right atrial pressure can be raised by the abdominal corset.

[14] DOCK, W.: Personal communication.
[15] BROWN, H.: Personal communication.

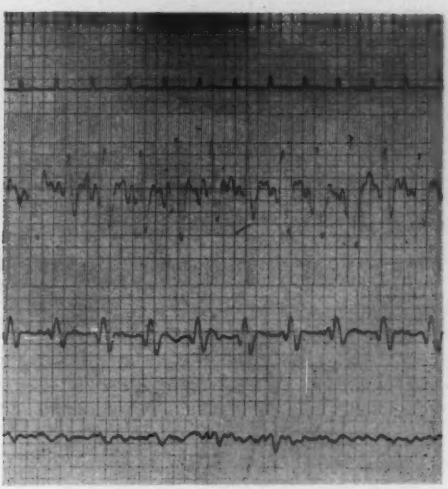

Figure 17.—Comparison of ballistocardiogram of patient with aortic insufficiency, normal person and patient with advanced coronary disease (reading from top to bottom).

The arrhythmias show bizarre patterns that can be well correlated with the abnormal physiologic findings (fig. 16). Patients with aortic insufficiency, anemia, beriberi, hepatic cirrhosis, and hyperthyroidism show large amplitude of the waves (fig. 17). Those with coarctation of the aorta show a characteristic shortening of the K

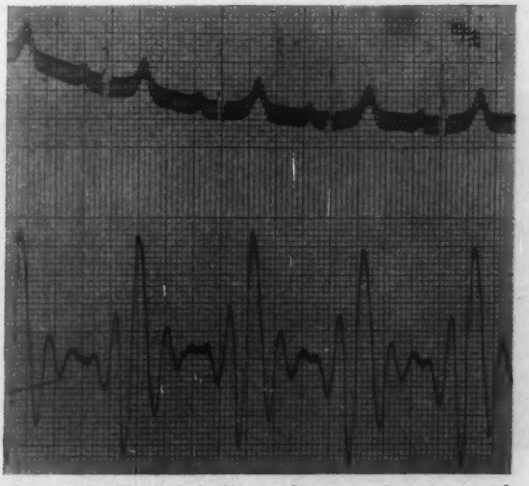

Figure 18.—Typical ballistocardiogram of patient with coarctation of the aorta. Note the short K wave.

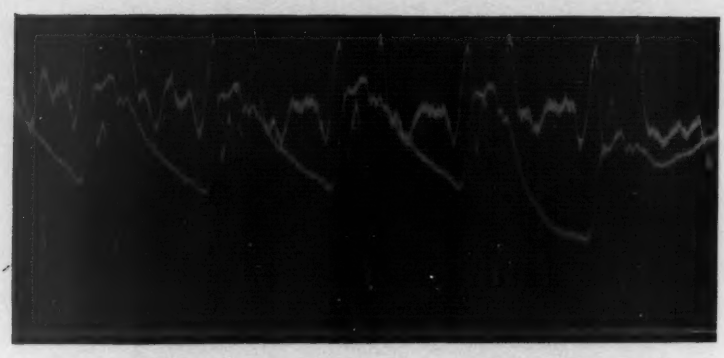

Figure 19.—Ballistocardiogram of a hypertensive patient showing the deep K wave and the notched J wave of early myocardial involvement.

wave (fig. 18). A short K wave believed to be caused by splanchnic vasoconstriction may be present in patients with severe hypotensive states such a shock. In fact this may allow a diagnosis of shock to be made earlier than by any other methods. A deep K wave characterizes the pattern seen in patients with hypertension or aortic rigidity. Hypertensive patients with early myocardial involvement show in addition a notched J wave (fig. 19). A deep K wave caused by the arteriovenous shunt of the placenta is seen in pregnant women; this disappears within 3 days after delivery. Large waves occurring in diastole are gallop waves or are caused by the high velocity of the narrow stream through a stenotic mitral valve (fig. 20).

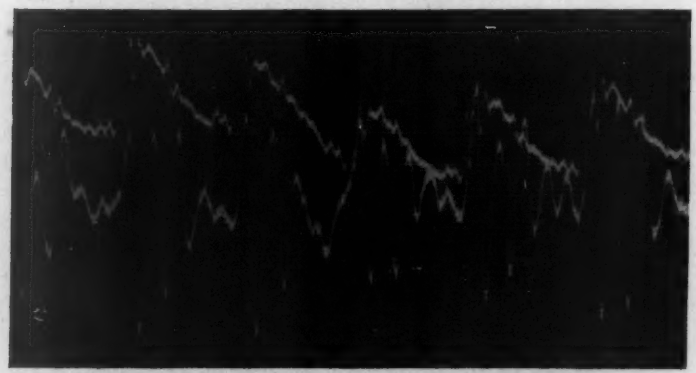

Figure 20.—Ballistocardiogram of a patient with aortic insufficiency in which large gallop waves are present in diastole.

PROGNOSTIC VALVE

The ballistocardiogram is of prognostic value clinically. A more accurate prognosis can be given in a case of myocardial infarction in which the ballistocardiogram returns to normal after the episode than in a case in which it remains abnormal. The same may be said

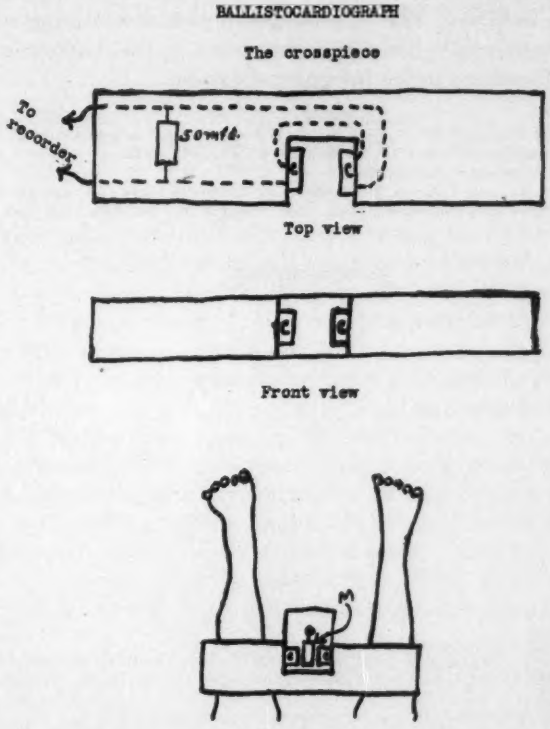

Figure 21.—Diagram of the slight change in construction of the ballistocardiograph employing two coils (C) *and a flat magnet* (M). *The magnet is fastened to a stand, whereas the crosspiece goes across the shins. The crosspiece is shown from the top and front views. The crosspiece and the magnet are shown in operating position.*

regarding such conditions as pericardial effusion, hypertension, and others. The change in the K wave in patients after operation for coarctation of the aorta may indicate the success of the operation. An abnormal ballistrocardiogram may indicate coronary artery disease

before any signs or symptoms appear.[11 14-17] The ballistocardiograph could in this way be of value in screening military personnel. Brown [15 18] has shown that cold will produce ballistocardiographic changes and make abnormal ballistocardiograms even worse.

Addendum.—Since this article was submitted for publication the electromagnetic type of ballistocardiograph has been modified. This is shown in figure 21 in which two coils (C) are employed with a flat magnet (M) mounted on a stand. The coils are wired so that the currents are additive. This allows ignoring exact centering of the magnet. The principle, however, is the same as in the electromagnetic instrument illustrated in the foregoing diagrams.

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[16] STARR, I., and WOOD, F. C.: Studies with ballistocardiograph in acute cardiac infarction and chronic angina pectoris. Am. Heart J. 25: 81–101, Jan. 1943.

[17] GUBNER, R.: Personal communication.

[18] BROWN, H. R., JR., and PEARSON, R.: Seasonal variations in heart and coronary disease as related to various environmental factors. Am. Heart J. 35: 763–768, May 1948.

A New Pickup for Ballistocardiography

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THE value of ballistocardiograms, which have been described as the records of the heart's recoil and the blood's impacts,[4] has only recently been realized by the medical profession. Although the pioneer work of Starr et al.,[5,6] and the later efforts of Hamilton et al.,[7] Nickerson,[8] and Brown et al.[9] has brought to light the diagnostic and prognostic qualities of these tracings, the equipment required has been of such size and expense as to discourage any but the research worker from using it. Recently, Dock,[10] has brought this type of recording within the reach of anyone who has an electrocardiograph. By the use of simple ingenious technics, using in one instance, a photocell and in another a magnet and coil, he has been able to record ballistocardiograms directly from the body. This will open a new field to the cardiologist and general practitioner as well, because the information derived from a ballistocardiograph is unobtainable by

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[4] STARR, I., and SCHROEDER, H. A.: Ballistocardiogram; normal standards, abnormalities commonly found in diseases of heart and circulation and their significance. J. Clin. Investigation 19: 437–450, May 1940.

[5] STARR, I.; RAWSON, A. J.; SCHROEDER, H. A.; and JOSEPH, N. R.: Studies on estimation of cardiac output in man, and of abnormalities in cardiac function, from heart's recoil and blood's impacts; ballistocardiogram. Am. J. Physiol. 127: 1–28, Aug. 1939.

[6] STARR, I., and MAYOCK, R. L.: On significance of abnormal forms of ballistocardiogram; study of 234 cases with 40 necropsies. Am. J. M. Sc. 215: 631–650, June 1948.

[7] HAMILTON, W. F.; DOW, P.; and RENNINGTON, J. W.: Relationship between cardiac ejection curve and ballistocardiographic forces. Am. J. Physiol. 144: 557–570, Sept. 1945.

[8] NICKERSON, J. L.: Some observations on ballistocardiographic pattern, with special reference to H and K waves. J. Clin. Investigation 28: 369–377, Mar. 1949.

[9] BROWN, H. R., JR.; HOFFMAN, M. J.; and DELALLA, V., JR.: Ballistocardiogram in coarctation of aorta; observations before and after operation. New England J. Med. 240: 715–718, May 5, 1949.

[10] DOCK, W., and TAUBMAN, F.: Some technics for recording ballistocardiogram directly from body. Am. J. Med. 7: 751–755, Dec. 1949.

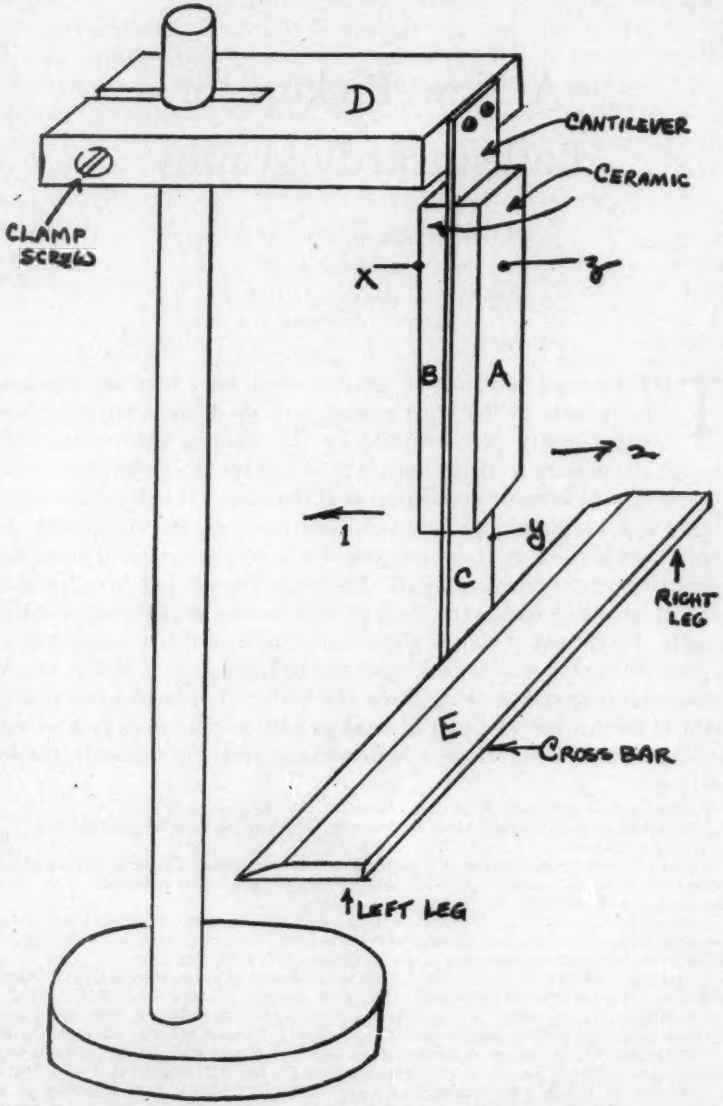

Figure 1.—Schematic diagram of pickup unit for ballistocardiography.

any other device or diagnostic aid. When these instruments are used, heart disease may be demonstrated in patients with angina and normal electrocardiograms and the work of Starr has shown its value in the asymptomatic patient. The frequent failure of the electrocardiograph to demonstrate coronary heart disease is well known, but the ballistocardiograph decreases the percent of patients with negative objective evidence.

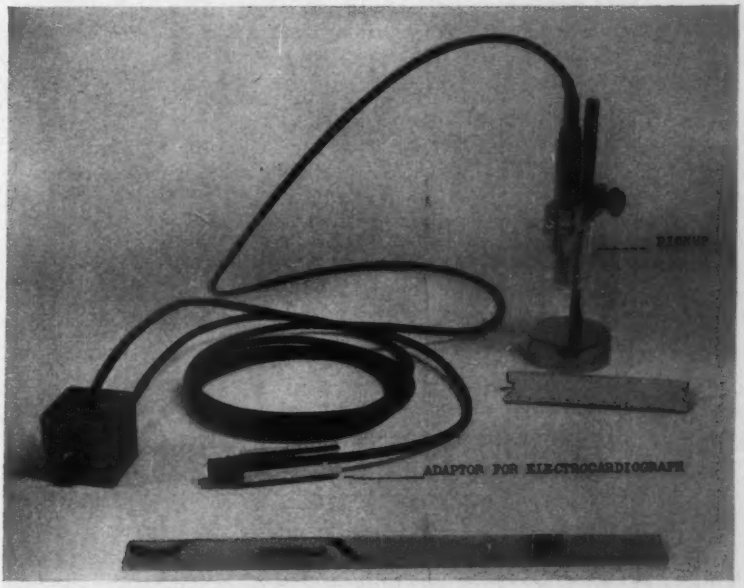

Figure 2.—Ballistocardiograph with shielded cable and adaptor for electrocardiograph.

While working with the photocell device, we sought to set up a wire elastic strain gage in its place. Further investigation produced the instrument which we wish to present here. We found that a pickup device using a piezoelectric system gave optimum results, and was eminently successful in recording ballistocardiograms directly from the body. The pickup unit is a self-generating device using a glennite [11] piezoelectric ceramic element (figs. 1, 2, 3, and 4). A piezoelectric material is, by its nature, one which when it receives a mechanical strain becomes electrically charged, and, hence, generates an electric potential. In this unit, piezoelectric plates A and B are mounted on each side of the metal strip C, which is held cantilever fashion to the

[11] Trade name of the Gulton Manufacturing Corporation.

base D. The patient lies supine on the table, and the crosspiece E is placed across the patient's bare legs and against the bottom of cantilever C, bearing a slight pressure against the latter.

The heart's recoil and the blood's impacts cause headward and footward displacements of the legs. When the patient's legs have a footward displacement, the cantilever is bent in direction "*1*" shown on the sketch. Element *A* is then put in tension and *B* is put in compression. These elements are so mounted that this causes positive voltage to be induced from *x* to *y* and from *y* to *z*. Hence, the voltages

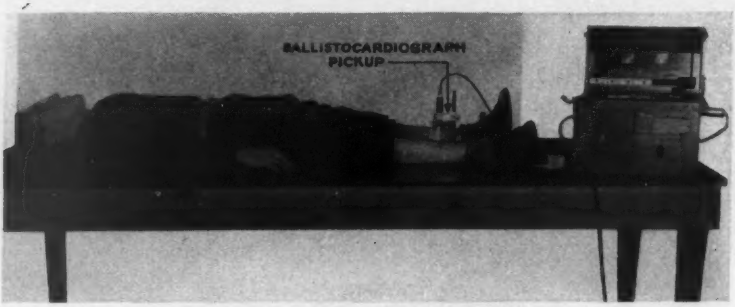

Figure 3.—Ballistocardiograph in use.

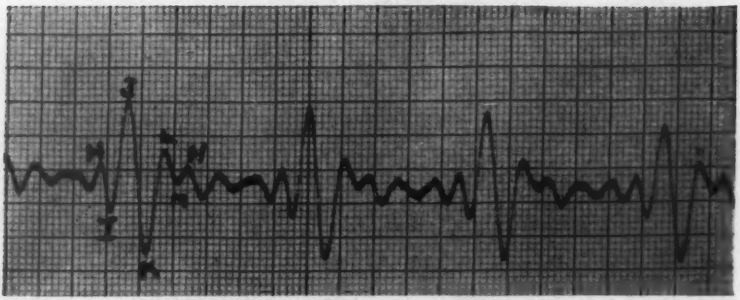

Figure 4.—Normal ballistocardiographic record obtained with new pickup.

are additive. Similarly, when the legs have a headward displacement, a voltage of the opposite polarity is induced and the cantilever is bent in direction "*2*." These voltages are then fed through proper electric networks, including amplifiers, and may then be recorded on any standard recorder. With this arrangement the magnitude of the needle displacement on the recorder is a measure of the displacement of the legs of the patient. Should one wish to have the recorder indicate the velocity of leg displacement, it is possible to introduce relatively simple electric networks which will make this conversion.

DISCUSSION

From our experience, the photocell device has been unsatisfactory because of difficulties with both the light source and the friction in the hinge. The magnet and coil device, described by Dock, measures velocity and there is some question yet as to whether the graph of velocity or of displacement will be of more clinical value. The present device measures displacement, and, by using the proper electric circuits, velocity or acceleration can be obtained. The electric output obtained from this new pickup element is 20 times that of the output obtained from a magnetic device, therefore ample voltage is available to operate any cardiograph or recorder without additional amplification. The excess voltage also makes it possible to use electric integrating or differentiating circuits to reveal hidden characteristics not easily recognizable in the original record. The records derived from this pickup are consistently reproducible, and obviate the need of frequent standardizations.

SUMMARY

Technics for recording ballistocardiograms directly from the body have recently been reported. A new pickup using a piezoelectric element is herein described. The electric output of the pickup is of such magnitude that various other circuits to record velocity or acceleration can be inserted without the need for amplification.

916061°—51——4

Long-Acting Heparin Preparation: A Useful Adjunct in Anticoagulant Therapy

A Clinical Trial of Depo-heparin Sodium in 15 Cases[1]

WILLIAM J. SMILES, *Lieutenant, junior grade, MC, U. S. N. R.*

A LONG-ACTING form of heparin, depo-heparin sodium [2] was tested on the Cardiovascular Service, U. S. Naval Hospital, Bethesda, Md., to ascertain its effectiveness and practicability as an anticoagulant and its usefulness as an adjunct to dicumarol in treating thromobo-embolic disease.

BREAKDOWN OF CASES

The drug was used in 15 consecutive patients in whom anticoagulant therapy seemed indicated. Nine were patients with coronary artery disease suspected of acute myocardial infarctions; 5 of these subsequently proved to have fresh infarcts, the other 4 had anginal attacks with severe transient myocardial ischemia. Five patients whose data went into this study had acute thrombophlebitis of the leg veins. Three of these had such extensive involvement as to be considered serious, one having already sustained a pulmonary embolism, while two who had more localized thrombophlebitis had shown some objective signs of extension prior to treatment. One patient suffered from an extensive deep thrombophlebitis; prior to anticoagulant therapy, he developed an acute myocardial infarction, and was promptly treated with heparin and dicumarol.

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[1] U. S. Naval Hospital, National Naval Medical Center, Bethesda, Md.

[2] Supplied in 1 cc. cartridges with disposable syringe. Each cc. contains:

Heparin sodium (20,000 units)	200 mg.
Gelatin	180 mg.
Dextrose	80 mg.

Preserved with sodium ethymercuri-thiosalicylate 1 : 10,000.

METHOD

The following routine was carried out as closely as possible: (1) An initial prothrombin time on both dilute and whole plasma was obtained. (2) A coagulation time by a modified Lee-White technic was determined. I did this personally in order to maintain standard procedure. (3) An initial dose of depo-heparin without vasoconstrictors was injected into the deep subcutaneous tissues, usually the anterior aspect of the thigh, and the area was marked. (4) In the absence of other factors such as chronic heart failure, extreme debility and cachexia, known liver disease, blood dyscrasia, or anticoagulant therapy prior to admission, dosages were based on weight. Patients who weighed from 100 to 160 lb. received an initial dose of 300 mg. (1½ cc). Those who weighed over 160 lb. were given 400 mg. (2 cc.). (5) In the next 24 hours the coagulation time was determined twice. (6) At the end of 24 hours a second prothrombin time was obtained. (7) Near the end of the first 24 hours a second dose of depo-heparin was injected in the opposite thigh and the site of the first injection was observed and palpated. This second dose of depo-heparin was either 200 or 300 mg., depending on the coagulation time near the end of the first 24 hours. (8) The patient was then started on dicumarol according to our usual schedule of 300 mg. the first, 200 mg. the second, and 100 mg. the third day. (9) On the second day of dicumarol therapy, a third dose of depo-heparin was given, again determined by the coagulation time; usually 200 mg. was sufficient. (10) As was anticipated, by the third day of dicumarol administration, the increase in prothrombin time had reached a satisfactory level of about twice normal, and if the coagulation time was then lengthened 2 or 3 times, no more depo-heparin was administered. The patient thereafter was maintained on a daily dose of dicumarol. (11) The results were carefully noted. (12) An autopsy was performed on the one patient who died with special attention to any evidence of subintimal or other hemorrhage as well as signs of complications of a thrombo-embolic nature.

RESULTS

The data was analyzed for: (1) the effect of depo-heparin on the coagulation time; (2) the apparent effect of depo-heparin on the prothrombin time during the first 24 hours with special reference to the dilute (1:4) plasma; (3) untoward reactions, local or systemic; and (4) clinical and autopsy results.

Effect on coagulation time.—In each patient a substantial and satisfactory increase in the coagulation time was observed following the initial dose of 300 or 400 mg. of the drug. One patient who received 200 mg. failed to show an adequate response.

Case report.—In a preliminary trial, one patient, a 67-year-old, 170-pound white man, had previously been somewhat sensitive to dicumarol. He received penicillin in oil and digitalis. In addition, he had a massive thrombophlebitis as well as a fresh myocardial infarction. He received 200 mg. of depo-heparin and simultaneously dicumarol therapy was restarted. He failed to show any significant response to the depo-heparin. This lack of effect on the coagulation time may have been because the initial dose was too small. The penicillin, the combined extensive thrombosis and the shocklike impairment of the patient's circulation all might have contributed to prevent a satisfactory anticoagulant effect. After 18 hours, a second dose was given with good results. This patient made a satisfactory recovery.

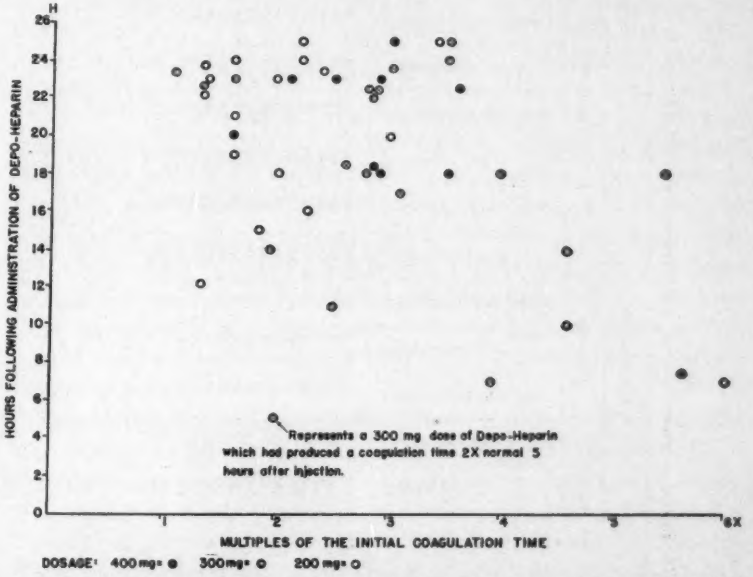

Figure 1.—Scattergram showing the coagulation times in multiples of the initial coagulation time plotted against the elapsed time after injection of a given dose of the depo-heparin.

A scattergram was prepared to show the levels obtained (fig. 1) in the first 24 hours, and for the entire period of depo-heparin administration until the second or third day of dicumarol therapy when the prothrombin time was about twice normal. It is observed that by using a smaller dose of depo-heparin on the second and third days there was no erratic change in the coagulation time after dicumarol therapy was started. The average initial coagulation time for these

TABLE 1.—*The initial coagulation time, prothrombin time and response to drugs in 15 patients.*

Case No.	Admission diagnosis	Initial base line							Subsequent dosage and therapeutic effect											
		First day							Second day				Third day				Fourth day			
		Weight (lb.)	Dicumarol (mg.)	Prothrombin time (sec.)	Depo-heparin (mg.)	Coagulation time (min.)	Depo-heparin (mg.)	Coagulation time (min.)	Dicumarol (mg.)	Prothrombin time (sec.)	Depo-heparin (mg.)	Coagulation time (min.)	Dicumarol (mg.)	Prothrombin time (sec.)	Depo-heparin (mg.)	Coagulation time (min.)	Dicumarol (mg.)	Prothrombin time (sec.)	Depo-heparin (mg.)	Coagulation time (min.)
*1	Myocardial infarction	163	0	14	0	16	300	40	300	19	200	50	200	22	200	32	200	31	‡	‡
2	Myocardial infarction	170	100	17	0	8	400	28	300	22	300	20	150	22	0	36	50	40	‡	‡
3	Thrombophlebitis	185	0	15	0	9	400	26	300	17	200	20	200	23	200	20	100	36	‡	‡
4	Thrombophlebitis	130	0	14	0	9½	300	23	300	18	200	30	200	17	200	18	50	33	‡	‡
5	Myocardial infarction	158	0	15	0	16	300	31	300	16	300	35	150	22	200	21	50	33	200	36
6	Myocardial infarction	180	300	18	0	7½	300	34	200	19	300	23	0	36	0	20	‡	‡	‡	‡
*7	Myocardial infarction	150	0	15	0	14	300	40	200	15	200	22	200	19	200	26	200	33	200	19
*8	Myocardial infarction	170	0	16	0	8½	400	48	200	17	300	30	200	20	200	58	200	24	200	27
9	Myocardial infarction	155	0	19	0	8	300	23	250	18	300	31	200	22	200	22	25	36	‡	‡
10	Thrombophlebitis	172	0	16	0	14	400	55	300	14	200	50	200	17	200	22	100	28	‡	‡
11	Myocardial infarction	147	300	13	0	11	300	23	200	20	250	29	100	32	0	35	50	28	‡	‡
12	Thrombophlebitis	178	300	15	0	14½	400	42	200	17	200	20	100	25	‡	‡	50	32	‡	‡
13	Thrombophlebitis+myocardial infarction	170	0	15	0	10	200	× 12	300	16	200	28	100	33	‡	‡	‡	‡	‡	‡
14	Thrombophlebitis	200	0	15	0	15	200	25	300	16	‡	‡	200	23	‡	‡	100	31	‡	‡
*15	Myocardial infarction	196	0	15	0	12	400	34	0	16	0	35	‡	‡	‡	‡	‡	‡	‡	‡

× See results, case report.
‡ Special record discontinued.
* Not substantiated.

patients was 10 minutes and 20 seconds at room temperatures of 25° to 26° C. (77° to 78.8° F.). Multiples of the original coagulation time up to 6 usually did not exceed 45 minutes and none were extended to 1 hour. Thus it can be seen from the scattergram that although many end-points are outside the arbitrary range of 2 to 3 times normal, all would seem to be within reasonable limits of safety. No hemorrhages occurred. The most noteworthy features appear to be: (*a*) Most of the coagulation times were 2 to 4 times normal and averaged 20 to 40 minutes. (*b*) Usually near the end of a 24-hour period a decrease in the coagulation time was noted. (*c*) The coagulation time of one patient increased 6 times with the initial dose of depo-heparin. He had been given dicumarol elsewhere but because red blood cells were found in his urine, the dicumarol therapy had been discontinued. The initial coagulation time was 7½ minutes, and the prolongation was still only 45 minutes in actual time. This patient's great activity and the consequent increased circulation apparently increased the rate of absorption from the depo-heparin deposit.[3] There was no return of the hematuria. This patient subsequently proved to have myocardial ischemia and evidence of an old infarct but apparently sustained no new injury on this occasion. (*d*) His coagulation time was permitted to fall below the arbitrary optimum range when it became evident that his prothrombin time was approaching the therapeutic level. (*e*) Table 1 which incorporates the essential data, shows that no difficulties were encountered in switching from depo-heparin to dicumarol. Three patients were started on dicumarol therapy immediately, and only two injections of depo-heparin were required.

Effect on prothrombin time.—Examination of the data for the first day of depo-heparin alone failed to reveal any significant change in the prothrombin time. Three records showed an average 10-second increase in the dilute plasma prothrombin time, a fourth showed a decrease of 10 seconds, the others showed practically no change. Whole plasma prothrombin time averaged about 1 second longer, which is within the range of probable error.

Untoward reactions.—No undesirable systemic effects were observed from any of the injections given (dosage ranged from 200 mg. to 400 mg.), but five mild local reactions occurred. One patient developed an erythematous elevated area of the skin over the injection site that resembled a giant urticaria in appearance and was slightly tender, but did not itch. Four patients complained of local pain 12 to 24 hours after the injection. Inadvertently one of these was

[3] BARCROFT, H., and EDHOLM, O. G.: Effect of temperature on blood flow and deep temperatures in human forearm. J. Physiol. 102: 5–20, June 30, 1943.

given intramuscularly. Another was in a hyperactive patient whose coagulation time was six times normal. He admitted having rubbed the area vigorously, which probably accounted for the pain. Objectively, some soft swelling was suggestive of a small extravasation of blood. Within 24 to 48 hours after the injection nothing was visible or palpable except the mark where the needle entered the skin.

Clinical results.—Of the 15 patients in this series, 14 recovered. One patient with a massive myocardial infarction entered the hospital on the fourth day following the onset of pain and was given depo-heparin which produced coagulation times in the "ideal" therapeutic range. He was subsequently satisfactorily maintained on dicumarol, but despite other supportive measures he developed ventricular tachycardia which did not respond to medical management and he died on the seventh hospital day. At autopsy the presence of a myocardial infarct was confirmed and evidence of congestive heart failure was found; no evidence of subintimal hemorrhage, hemorrhagic phenomena, or embolic phenomena were found.

In another patient an extension of a posterior apical infarction occurred on the sixteenth day after the original infarct, 12 days after depo-heparin was discontinued, when the prothrombin time suddenly dropped to 23 seconds (normal 15). The anticoagulant effect of depo-heparin had been entirely within the therapeutic range. This patient had been persistently overactive from the beginning, refused to use the oxygen provided, and continued to smoke. After the second episode he was more cooperative and made a good recovery.

The patients with thrombophlebitis improved without signs of further extension or emboli and with little or no residual damage. While statistically not significant, the results from this small series of patients compare well with statistics available from extensive composite reports dealing with the use of anticoagulants.[4]

SUMMARY

Depo-heparin without vasoconstrictors can be used effectively as an adjunct to dicumarol therapy. A suitable check on the therapeutic levels attained is the modified Lee-White method of determining the coagulation time which will give a normal average end-point between 5 and 15 minutes. Making this determination once a day near the end of the 24-hour period following injection should be sufficient for the average patient. The administration of depo-heparin without vasoconstrictors in doses of 300 to 400 mg. initially and 200 to 300 mg. given 23 to 24 hours later on successive days gives an increase in coagulation

[4] WRIGHT, I. S.: Use of anticoagulants in treatment of diseases of heart and blood vessels. Ann. Int. Med. 30: 80, 1949.

time of 2 to 4 times normal. A single injection of this dosage will give a lengthened coagulation time of 2 to 4 times normal for about 24 hours. No complications were encountered either in control or management while combining depo-heparin with dicumarol and the switch-over was accomplished with ease. The prothrombin time was not appreciably changed by depo-heparin during the first 24 hours when it alone was used. The effect of dicumarol was reflected by the increased prothrombin time for all practical purposes as if it were being used alone. No significant discomfort follows the use of depo-heparin provided it is injected at body temperature into the deep subcutaneous tissues. This is superior to intramuscular injection. To obtain optimal results, the patient should be cautioned not to apply pressure at the site of injection.

CONCLUSIONS

A satisfactory anticoagulant effect was produced during the first 24 hours by a single adequate injection of depo-heparin. The trend of opinion among the medical officers who observed these patients is that depo-heparin is a satisfactory adjunct when used with dicumarol.

A Pressure Method of Investment

PIERRE O. EVANS, *Lieutenant Colonel, DC, U. S. A.*[1]

THE problem of obtaining a "bubble-free" investment of wax patterns and stone dies has vexed the dental profession ever since the precision casting of wax models was developed. Many aids to the alleviation of this trouble such as vibrators and surface tension reducers have appeared and have helped but the percentage of gold spurs and nodules present still appears high in the finished castings as performed by the dentists and technicians who do not use auxiliary mechanical apparatus. Although the use of negative pressure (vacuum) investment machines gives highly satisfactory results, the cost of a commercial vacuum machine is usually beyond the means or needs of the average general practitioner and is not always obtainable for the average Army dental clinic. Although a vacuum apparatus can be improvised by using the water column from a sink faucet, in many cases the tap water pressure is insufficient to create enough negative pressure in the short span of time needed to dissipate the air bubbles next to the pattern before primary crystallization of the investment begins.

In contrast to the negative pressure technic of removing air from the investment, at this hospital a positive pressure device is used. The theory underlying this type of investment is that pressure applied will be equal in all directions and the air will be reduced in volume by the square of the number of atmospheres of pressure applied. From a clinical viewpoint it must be admitted that even with the most careful investing technic, air in the form of bubbles will be present in the investment, but by applying pressure the size of the bubbles will be decreased to the point at which they will produce negligible end results. The pressure device is of simple construction, consisting of an air valve, gage, a 6-inch length of pipe with a 3-inch diameter, and two threaded pipe caps, one of which has the air gage threaded through it (fig. 1). Artificial stone is used to build up the inside of the lower cap to reduce the number of turns necessary to effect closure and to form a base for the rubber gasket.

[1] Madigan Army Hospital, Tacoma, Wash.

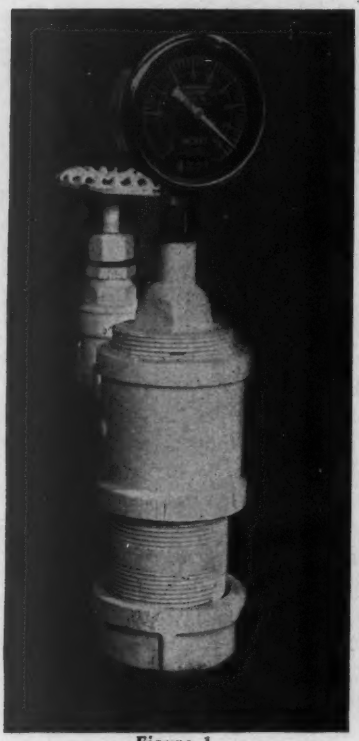

Figure 1.

Figure 2.

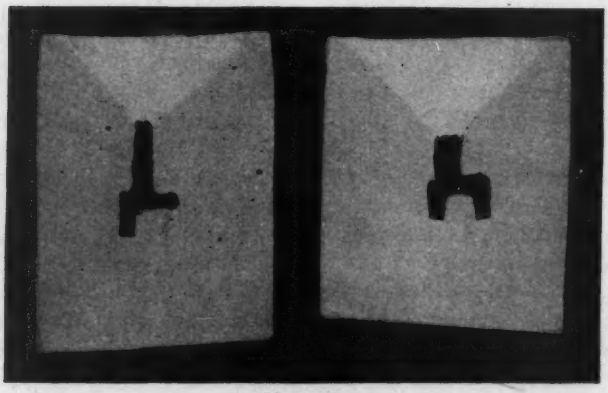

Figure 3.

Investing for this type of device consists of (1) painting the sprued wax pattern with investment, (2) placing the ring on the sprue base and sealing the ring to the base with red utility wax by hand pressure to stabilize the ring for handling, (3) filling the ring with investment, (4) placing it on the lower cap (fig. 2), (5) screwing the lower cap to place and advancing the air pressure to the number of atmospheres desired, and (6) maintaining this air pressure for the period of investment set which can be determined by leaving out a test daub.

Figure 3 illustrates the potentialities of this type of investing. Both patterns were invested from the same investment mixture. The mold on the left was painted and then vibrated moderately and allowed to set; the mold on the right was merely poured and subjected to pressure for 15 minutes. This pressure method is also used for pouring stone dies for crowns and inlays made by the indirect method. The dies obtained by pressurizing are not only free from bubbles, but seem to be more dense and, as a consequence, less liable to marring and chipping. One hundred inlays and crowns have been invested by this technic, and in no instance were spurs or nodules present on the castings.

CONCLUSIONS

The results obtained thus far with the pressure investing technic are more than satisfactory. Costs of construction of the chamber are negligible because scrap and salvage materials can be used. The time element as compared to other technics is not increased. In a preliminary study of 100 investments under pressure, none showed faults caused by bubbles in the investment. Forty-five pounds (about 3 atmospheres) of air pressure is used at this station. In accordance with the theory used, the air bubbles present will be compressed to one-ninth their original size, thus presenting a minimum of faults.

An Electric Defibrillator for Cardiac Resuscitation

JOHN H. STOVER, JR., *Lieutenant Commander, MC, U. S. N.*[1]

RECENTLY Johnson and Kirby [2] reported a series of successful cardiac resuscitations using an electric current of moderately high amperage applied directly to the heart. I have designed and constructed a defibrillator to replace their original experimental apparatus. Because ventricular fibrillation is a matter of great concern to surgeons, it is believed that a description of the device may be of general interest.

Design considerations.—The apparatus (1) should deliver 2 amperes on short-circuit test of the electrodes,[3] (2) must be safe, and (3) must be reliable.

General description.—The defibrillator consists essentially of an isolating transformer, a current limiting resistor, and a reliable fast-acting electric switch. It is also reassuring to the surgeon to read a meter before attempting cardiac defibrillation. In my apparatus, additional testing circuits are included; these are intended primarily for convenience in animal experimentation, when it may be desired to change the current. The circuit diagram (fig. 1) shows the basic circuit in heavy lines and the auxiliary test circuits in light lines.

TECHNICAL DESIGN AND CONSTRUCTION FEATURES [4]

Power.—The values and power ratings indicated in the list of components will reliably supply 2-ampere pulses.

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[1] The Johnson Foundation for Medical Physics, University of Pennsylvania, Philadelphia, Pa., at the time this article was written; now with the Department of Biophysics, the Johns Hopkins University, Baltimore, Md.

[2] JOHNSON, J., and KIRBY, C. K.: Cardiac resuscitation. S. Clin. North America 29: 1745–1753, Dec. 1949.

[3] JOHNSON, J., and KIRBY, C. K.: Personal communication.

[4] If the defibrillator is to be used in a cyclopropane atmosphere, it should be completely sealed in a shielding can, or gasproof switches should be employed.

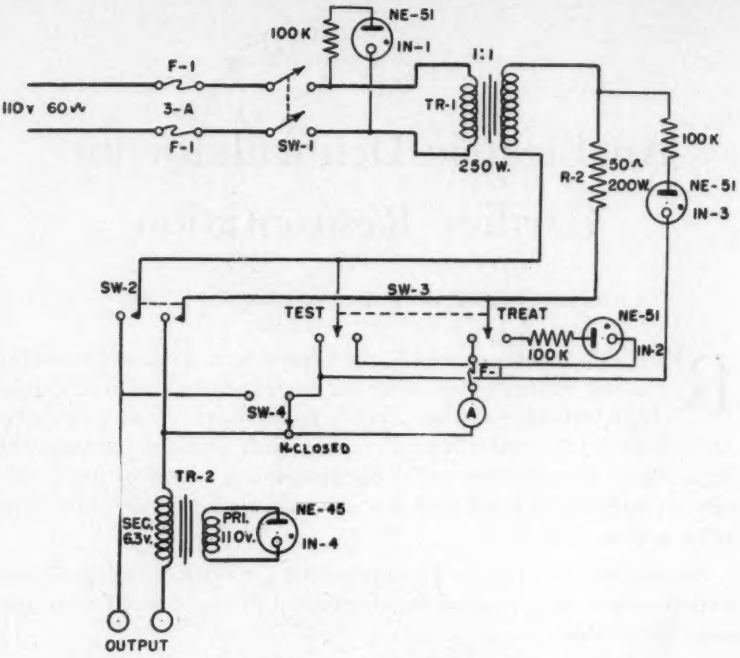


Figure 1.—Schematic circuit diagram.

Safety features were a major concern.

(1) Isolating transformer.—It is possible to obtain 2 amperes directly from the house current through a limiting resistor. However, since one side of the line is customarily grounded, one of the electrodes is "hot." In many operating-room suites, the patient is intentionally grounded, and since the surgeon may also be grounded (wet floor, grounded grid in floor), a direct path is provided for the flow of a potentially lethal electric current. This was a major objection to the original Johnson-Kirby apparatus. The condition was corrected by employing an adequately rated "isolating" transformer. With the transformer in the circuit, neither electrode is "hot" with respect to ground. The patient may be grounded or not as desired, and the surgeon does not have to worry about an electrical ground because of a torn glove.

(2) Defibrillator switch.—Because the heating effects of a 2-ampere current are great, the shock must be of short duration—about 0.1 sec. Consideration was given to electronic and mechanical methods of automatic cut-off after 0.1 sec. Although a ruggedly constructed

"micro" push-type switch, with a telegraph key button, a warning sign reading "strike and release fast," and a large indicator jewel that glows during the flow of the defibrillating current (fig. 2) is satisfactory, an automatic timer would be desirable if personnel not familiar with the apparatus are required to operate it.

(3) Electrode current.—The device is so arranged that the electrodes are always "dead" unless the operator is actively maintaining pressure against either the "defibrillate" switch, or simultaneously maintaining pressure against the "selector" and "electrode test" switches. Whenever the electrodes are conducting current, the large jewel glows. To accomplish this, a small filament transformer is used to provide a surge to fire the neon lamp.

(4) Fuses.—Both sides of the line and the meter are fused at 3 amperes. Small panel-mounting fuses are used.

(5) Binding posts should be of high quality and completely insulated.

(6) Test circuit safety features.—To preclude the possibility of the selector being set on "test" when the surgeon desires to defibrillate, the indicator jewels flash—yellow for "test," and red for "treat." In addition, the selector is a toggle switch with a spring return from one side, i. e., pressure must be maintained against it for "test," but for "treat" it is merely flipped to the other side. For testing the continuity of the electrode and the electric cord the selector switch must be

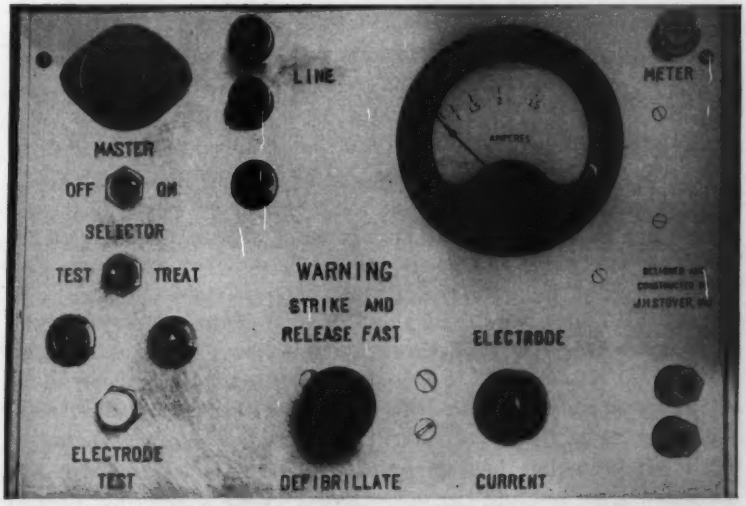


Figure 2.—Front view of panel: electrodes not connected.

916061°—51——5

set at "test," and the "electrode test" push button must be depressed and held.

RELIABILITY FEATURES

High quality components are used throughout.

Neon indicator lamps.—These are long-lived, quick-lighting, and stand up under mechanical shocks better than filament type lamps. More important, failure of a neon lamp does not result in a change in the output of the defibrillator, but failure of a filament type would. The alternative of using low-voltage filament bulbs involves an additional transformer and switch contacts, and hence more potential failure points.

Connections are soldered throughout. If direct connections are used, lock washers should be inserted.

ELECTRODES

The electrodes used by Johnson and Kirby were oval copper plates measuring about 2 by 3 inches, and soft enough to be molded about the heart. A high quality, rubber-covered electric cord was soldered to the electrodes, and pin tips were soldered to the defibrillator end of the cord for insertion in the binding posts of the defibrillator. Such a cord has withstood weekly autoclaving for a year. If cold sterilization is used, a conventional outlet and plug can be employed. At present we are testing a simple two-conductor "banana" plug (which the binding posts we employed also accept).

OPERATION

Basic circuit type.—Plug in to 60-cycle line. Switch on. Connect electrodes, and bring them in contact with one another. Depressing defibrillator switch should cause large jewel to glow. To defibrillate, place on heart and give defibrillator switch a quick, glancing blow.

Basic plus test circuit type.—Plug in to 60-cycle line. Switch on. When selector switch is held on "test," the meter will indicate the amperage that the device will supply on short circuit of electrodes. To check electrodes and cords for continuity, connect electrodes, bring in contact, hold selector on "test," and depress "electrode test" switch. The large jewel will glow, and the meter will indicate the amperage actually flowing through electrodes. This method may also be used for determining the current flowing through heart of an experimental animal. For defibrillation set the selector on "treat" and when ready, strike the defibrillator button as before. The meter will *not* indicate, but the large jewel will flash. This is to discourage operators from holding the defibrillator button down long enough to "get a reading" at the expense of the patient's ventricular muscle.

The small "test" and "treat" indicators may blink alternately if cardiac resistance is low.

LIST OF COMPONENTS

Basic type

TR–1	One isolating transformer 115 V, 1:1, 250 watt (Stancor type P 6161[5]).
R–2	One voltage divider ceramic-type resistor, 50 ohm 200 watt.
SW–1	One double-pole, single-throw line switch, 3 ampere, 115 volt.
SW–2	One defibrillator push switch—double-pole, single throw. Must be spring return to open position (Micro switch, Type DB.RH1 Serial No. A, rugged, smooth acting, shaft is tapped so large telegraph key button may be used. Terminals 5 and 6 are not used).
IN–1	One panel indicator light assembly for type Ne 51 neon bulb (red, amber, or clear only). Some assemblies have built-in resistor, if not, a 100,000 ohm ½-watt series resistor (R–1) must be used.
IN–4	One panel indicator light assembly, large (1-inch) for type Ne 45 neon bulb (red, amber, or clear only). No dropping resistor necessary. A small assembly and Ne 51 bulb plus 100,000 ohm ½-watt resistor may be used here.
TR–2	One filament transformer—6.3 volts or 2.5 volts.
F–1	Two fuse holders and two 3-ampere fuses (such as size "3 AG").
	Electrodes, electrode cord, and connectors.
	Line cord.
	Case and panel or "bread board."

Additional parts for test circuit

AM	One radio frequency or alternating current ammeter, 0–3 amperes, panel mounting, 3-inch.
F–1	One fuse mount and 3-ampere fuse (for meter protection).
SW–3	One double pole double throw three-position toggle switch with spring return from one "on' position to "off" position 3 amperes 110 volts.
SW–4	One single pole double throw push button switch normally closed, spring return from open position (standard "micro" switch).
IN–2, IN–3	Two small indicator lamp assemblies and 100,000 ohm ½-watt resistors (R–1) for Ne 51 neon bulbs—one red, one clear or amber jewel.

[5] Stancor isolating transformer is supplied with built-in line cord. In addition, it has a switch for setting to local voltage. If a basic type defibrillator is constructed, an AC ammeter should be connected across output of defibrillator, and a transformer switch should be set to position which most nearly gives 2 amperes. With a line voltage of 115, these taps give 2.5, 2.3, and 2.1 amperes, respectively.

Arterial Blood Culture

MELVIN B. SULLIVAN, JR., *Lieutenant, junior grade, MC, U. S. N. R.*[1]
CLIFFORD P. POWELL, *Captain, MC, U. S. N.*[1]

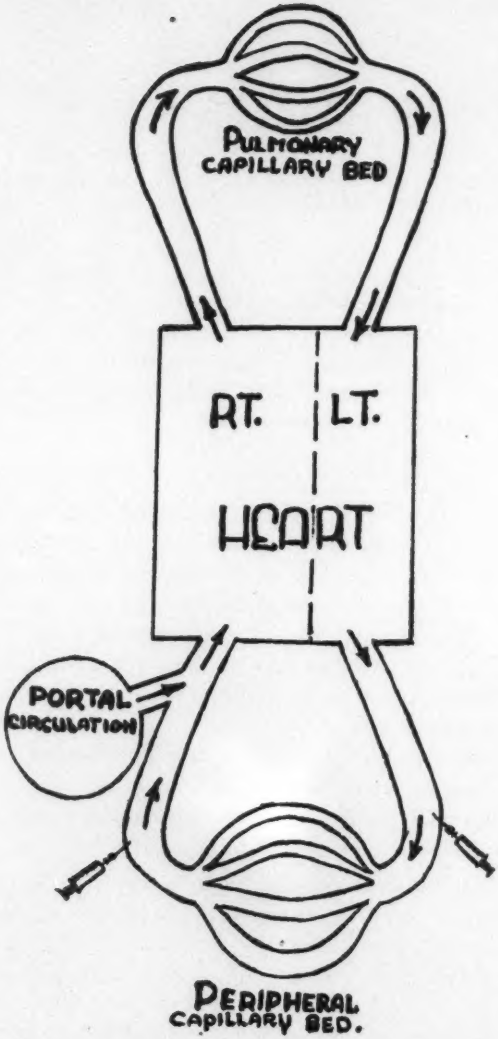
WITH the advent of new and specific antibiotics for the treatment of blood-borne infections, it is essential to determine the exact etiologic agent and its sensitivity to the various antibiotics with a minimum of delay. With this in mind, it is considered advantageous to use the described technic for arterial blood cultures as an adjunct to routine venous blood cultures.

CASE REPORT

A patient with most of the clinical findings of subacute bacterial endocarditis, but with negative venous blood cultures, was transferred to this hospital after unsuccessful treatment with penicillin and caronamide. In view of the previous therapeutic failure and the long-term therapy likely, it was deemed urgent to have the infecting organism isolated and its sensitivity to the antibiotics determined. At the suggestion of a civilian consultant[2] an arterial blood culture was taken. It was positive for *Streptococcus viridans*, while the concomitant venous culture was negative, making possible the sensitivity studies which demonstrated the need for large doses of penicillin (12,000,000 units daily). This therapy effected an apparent cure without any evidence of a remission 9 months later.

The rationale for the procedure is shown in figure 1. The primary sites for blood stream infection are through the peripheral circulation, the portal circulation, the pulmonary circulation, and the heart. The rationale for proposing the arterial culture as a more accurate means of diagnosing blood-borne infections is theoretical. There may be a filtering action exerted on the bacteria as they pass through the pulmonary and peripheral capillary network at a reduced speed. That such a phenomenon exists is suggested by the peripheral petechiae

[1] U. S. Naval Hospital, Philadelphia, Pa.
[2] DURANT, T. M.: Personal communication.



FILTRATION DIAGRAM

Figure 1.

seen in subacute bacterial endocarditis, and by the comparative increase in positive arterial blood cultures as compared to simultaneously drawn venous blood cultures. Murray and Moosnick[3] reported a comparative study on 27 cases of possible septicemia in which 15 were proved by blood cultures. In this group, there were 6 positive venous blood cultures with one negative companion arterial culture. In the same group, 14 positive arterial blood cultures were obtained with 9 negative companion venous cultures. This represented an increase of 133.3 percent in accuracy of diagnosis; that is, arterial cultures were 2⅓ times more effective.

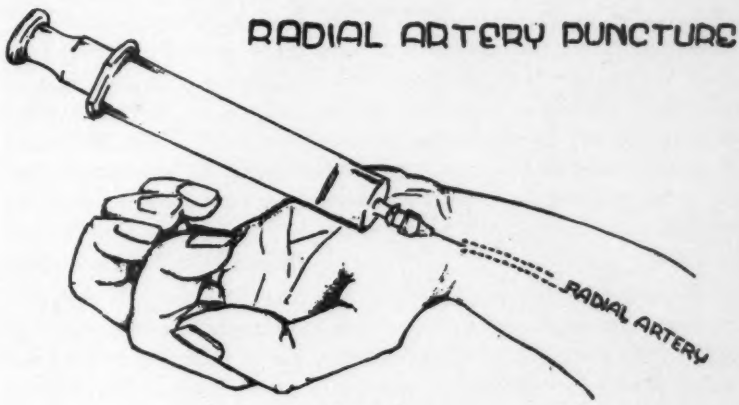


Figure 2.

The radial artery has proved to be easily located and punctured without causing excessive pain. The technic in brief is to cleanse the area over the artery at the wrist with iodine and using a 21-gage needle, enter the skin at about a 35-degree angle (fig. 2). If pressure is released for a moment after the needle penetrates the skin, the artery will tend to roll back under the needle and minimize probing. No tourniquet is necessary, and there is not a vein of sufficient caliber to enter in this area. If the radial artery is not easily palpable, the femoral artery is a satisfactory substitute. Usually, the pain is not of sufficient magnitude to require an anesthetic and momentary local pressure is adequate for hemostasis.

It is believed that this procedure has a definite place in our diagnostic armamentarium although as yet it has not been adequately evaluated. Our facilities prohibit a prolonged simultaneous com-

[3] MURRAY, M., and MOOSNICK, F.: Simultaneous arterial and venous blood cultures. J. Lab. & Clin. Med. 26: 382, Nov. 1940.

parison of the venous and arterial blood cultures, and it is hoped that this article will stimulate such a study.

CONCLUSIONS

The need for early determination of the specific infecting organism and its sensitivity to the antibiotics has become important with the development of a variety of new antibacterial agents. The relatively unused arterial blood culture is suggested as an improved means of obtaining this information at the earliest possible time.

The Dynamics of Lumbar Puncture

GORDON T. WANNAMAKER, *Captain, MC, U. S. A.*[1]

SOME of the most elementary principles of physics are often overlooked in performing a lumbar puncture. The spinal tap, consisting of inserting a needle into the subarachnoid space of the lumbar region for the purpose of determining the spinal fluid pressure or obtaining spinal fluid for laboratory examination for diagnostic purposes, is a relatively simple but occasionally dangerous procedure, the danger being caused by failure of the operator to comprehend the dynamics involved and to avoid sudden changes in pressure. Spinal taps performed for suspected increase in intracranial pressure and spinal cord lesions producing blocks require moderately different technics.

Spinal taps can be performed safely on any patient suspected of having increased intracranial pressure or a tumor in the posterior fossa provided there is no sudden change in the dynamics. This necessitates the employment of a closed system throughout the entire procedure with gradual changes of pressure. The tap should be performed with the patient lying on his side and never in the sitting position. Under no conditions should the needle be inserted into the subarachnoid space, the stylet removed, and the spinal fluid allowed to spurt freely while the manometer is being attached. It is well to remember in performing the spinal tap that one is dealing with fluid under pressure in a closed system and pressure changes in one part are reflected throughout the entire system.[2] In the presence of increased intracranial pressure a sudden release of pressure from below may cause one of two serious complications: (*a*) slight downward displacement or herniation of the contents of the posterior fossa of the cranial vault, and (*b*) herniation of the hippocampal gyrus through the tentorial notch compressing the midbrain.[3] Herniation

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[1] Walter Reed Army Hospital, Washington, D. C.

[2] MASSERMAN, J. H.: Cerebrospinal hydrodynamics; clinical experimental studies. Arch. Neurol. & Psychiat. 32: 523–553, Sept. 1934.

[3] REID, W. L.: Lumbar, ventricular and cisternal puncture; their indications and their dangers. M. J. Australia 1: 311–317, April 10, 1943.

of the cerebellar tonsils and medulla through the foramen magnum may cause death either by severe damage to the vital centers of the medulla or by increasing congestion of an already poorly compensated brain.

The following procedure is recommended to prevent sudden changes in the spinal fluid pressure, to allow one to record safely the pressure, and to obtain sufficient fluid for laboratory tests. With the patient lying in the lateral recumbent position, under local procaine anesthesia, a No. 18 gage spinal needle is inserted into the interspace be-

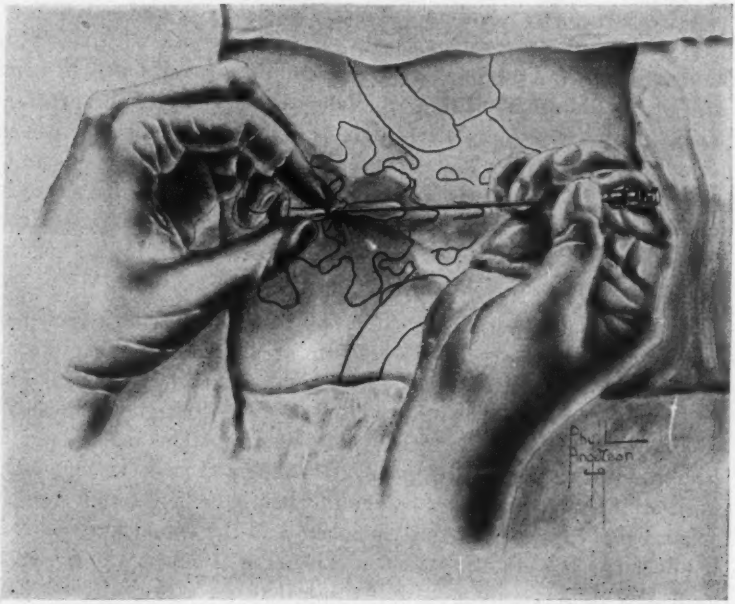
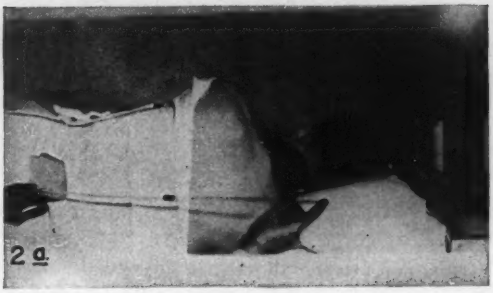


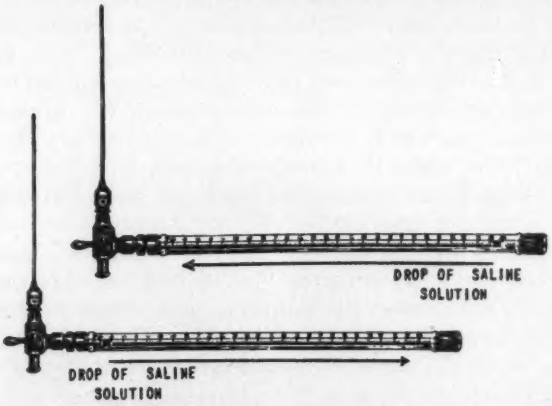
Figure 1.—Showing tip of spinous process of the fourth lumbar vertebra held between thumb and index finger, left hand, with insertion of 18-gage spinal needle between spinous processes of the fourth and fifth lumbar vertebrae.

tween the fourth and fifth lumbar vertebrae which usually lies on a line drawn through the iliac crest. To facilitate insertion of the needle in the midline it is helpful to grasp the lower tip of the spinous process of the fourth lumbar vertebra between the index finger and thumb (fig. 1), and to insert the needle just below this point, in the usual manner, down to the interlaminar space where it will meet resistance at the ligamentum flavum. At this point the stylet is withdrawn and the water manometer attached horizontally to the needle. A small drop of sterile saline solution is placed just below the junction of the tubes of the manometer. The three-way stopcock is opened

between the needle and the manometer (fig. 2a). The needle with the manometer attached is then pushed in further with the left hand, the right hand supporting the manometer. As the point of the needle passes through the ligamentum flavum into the epidural space the drop of fluid in the manometer flows toward the needle showing an area of negative pressure. The index finger of the right hand is then placed over the free end of the manometer to act as a ball valve so that pressure within the manometer can be controlled. The needle is then inserted into the subarachnoid space. At this point the drop of fluid in the manometer will start to flow away from the needle as the first drop of spinal fluid enters the needle and before spinal fluid is visible in the manometer. The manometer is then rotated to the vertical position (fig. 2b). Using the right index finger as a valve over the open end of the manometer the drop of saline solution is allowed to rise slowly until the spinal fluid is apparent in the manometer, and similarly the spinal fluid is allowed to rise at the rate of 1 cm. per second until the initial fluid pressure is obtained. Thus there is no sudden change in the dynamics of the spinal fluid and the pressure is obtained without danger to the patient. As a check on the accuracy of the obtained pressure one must make sure there is no block at the point of the needle by noting the slight fluctuation of pressure in the manometer synchronous with respiration and cardiac pulsation. The fluid in the manometer is then drained into a test tube by means of the stopcock. Each segment of the manometer contains about 1 cc. Similarly the manometer is again filled, pressure obtained, and fluid collected in the test tube. From 2 to 3 cc. of fluid are usually sufficient for total protein determination and the cell count. The stopcock is turned off and the needle quickly withdrawn. The Queckenstedt test is contraindicated in the presence of increased intracranial pressure as it further increases the pressure from above, forcing the brain down, and may result in death or serious complications.

In suspected complete or partial spinal canal blockage, lumbar puncture is of great diagnostic aid if properly performed. The method employed should be standardized so that the results obtained can be easily duplicated by other observers as is often necessary in the Army chain of evacuation. Furthermore, the procedure employed should allow one (*a*) to follow the changing degree of block of an expanding or subsiding lesion by performing serial tests, or (*b*) to check the effects of therapy instituted to relieve the cause of block. The needle is inserted below the level of the suspected block, usually between the fifth lumbar and the first sacral vertebrae. An 18-gage spinal needle should be used because a needle of smaller bore results in delayed pressure readings. The closed system of inserting the needle and obtaining the initial pressure as previously described is de-





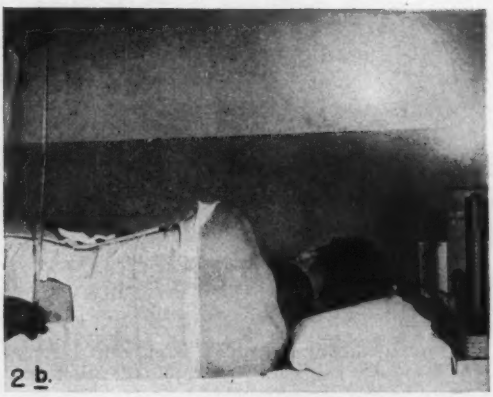


Figure 2.—Showing closed technic employing drop of saline solution as an indicator for localization of tip of spinal needle. The drop going down in the epidural space showing negative pressure and up in the subarachnoid space showing positive pressure. Arrows indicate direction drop will travel from original position shown.

sirable. An initial loss of a few cubic centimeters of spinal fluid in the presence of a complete block in the spinal canal may so reduce the pressure below the block that fluid does not rise in the spinal fluid manometer, particularly if the tap is being performed in the prone position, in which the fluid must rise vertically through the length of the spinal needle before the column of fluid becomes visible in the manometer.

After the initial pressure is obtained, it is ascertained that there is no blockage of flow of fluid at the spinal needle point; such blockage may be caused by a nerve root lying adjacent to the needle aperture acting as a valve. Such an obstruction of the needle would give inaccurate results in relation to changes of fluid pressure within the subarachnoid space. If there is no obstruction of the needle there will be changes in spinal fluid pressure with cardiac pulsation, deep inspiration and expiration, sneezing, coughing, and straining. Such changes are present even below the site of complete block. This does not mean that there is free communication of flow of spinal fluid from the cranial vault down to the site of insertion of the spinal needle. Such changes merely reflect changes of venous pressure within the spinal canal.

Thus assured of a patent needle, the initial pressure of spinal fluid in millimeters of water is recorded. The cuff of a sphygmomanometer is wrapped about the patient's neck (fig. 2), explaining the reason for this to the patient. A bilateral Queckenstedt test is performed by having an assistant pump up the sphygmomanometer cuff, 10 mm. of mercury each 10 seconds until 40 mm. of mercury is reached. The spinal fluid pressure is recorded at the completion of each 10 seconds. Then at 10-second intervals the sphygmomanometer pressure is lowered 10 mm. of mercury until zero mm. pressure of the cuff is obtained, and the spinal fluid pressures are recorded (fig. 3).

Normally the pressure will rise rapidly and fall at the same rate. In a partial obstruction the rising component of the curve will be flat at first, then as the increase in pressure above the block becomes great enough to force fluid past the partial obstruction there will be a more rapid rise. There is a direct relationship between the amount of pressure required from above (i. e., in the sphygmomanometer cuff) to force fluid past the partial block and the degree of block. After release of the cuff pressure and thus the pressure of spinal fluid above the lesion, there is a much slower reduction of pressure below the lesion, so that a flattened or slowly falling curve is produced. The length of time required for the pressure to return to the initial level is again in direct proportion to the degree of partial block, often taking 3 or 4 minutes.

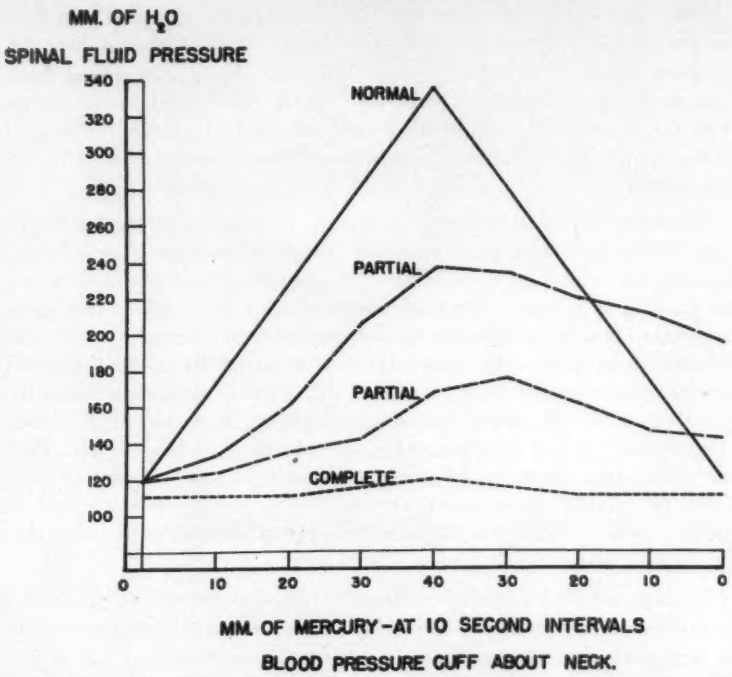


Figure 3.—Graphic representation of results obtained by a quantative Queckenstedt test for spinal subarachnoid block in patients with complete block, partial block, and absence of block (normal).

In complete block there is little or no change in the spinal fluid pressure below the site of the lesion on applying the Queckenstedt test. In a complete but somewhat elastic block there may be a slight rise in spinal fluid pressure when the Queckenstedt pressure is from 30 to 40 mm. of mercury. This pressure will usually fall rapidly when the cuff pressure is reduced below 30 mm. After removal of 2 or 3 cc. of spinal fluid in a complete block, the spinal fluid pressure will approach zero mm., and removal of from 5 to 10 cc. depending on the total volume of fluid below the site of the complete block, will produce a negative pressure.

By an accurate determination of spinal fluid pressure, using the afore-mentioned method, one can diagnose and follow the progress of lesions producing complete and partial spinal canal block. This method, a modification of the technic described by Grant and Cone,[4] has been used to great advantage in evaluating back injury and po-

[4] GRANT, W. T., and CONE, W. V.: Graduated jugular compression in lumbar manometric test for spinal subarachnoid block. Arch. Neurol. & Psychiat. 32: 1194–1201, Dec. 1934.

tentially paraplegic patients at this center. In several patients a partial block has been reduced by hyperextension and surgical decompression has thus been avoided. Other patients in whom a partial block showed signs of increasing were decompressed surgically with excellent postoperative results.

SUMMARY

The closed system for performing lumbar puncture is safe and avoids sudden changes of pressure in all patients in whom increased intracranial pressure is suspected. When properly performed the closed system enables the operator to obtain valuable information about spinal fluid pressure, cell count, and chemistry. Graduated jugular compression in the lumbar manometric test for spinal subarachnoid block is of particular value in following patients with back injuries and paraplegia. Standardization of this procedure at all medical installations along the chain of evacuation to a neurosurgical center would be of great value in determining the progress of the lesion, formulating its prognosis, and instituting proper therapy.

Myocardial Infarction in Young Adults

MASON R. BAKER, *Captain, U. S. A. F. (MC)* [1]
WILLIAM R. SCHILLHAMMER, JR., *Captain, MC, U. S. A.*[1]

MYOCARDIAL infarction in young adults is seldom seen by the practicing physician. The following cases were studied at this hospital.

CASE REPORTS

Case 1.—A 22-year-old soldier had been in excellent health except for occasional pain in the left anterior side of his chest until 2 days prior to admission to the hospital. At that time he suffered a severe substernal pain which lasted only a few seconds. This soon disappeared and he felt well until that evening when, while sitting on his bed in his barracks, he suddenly felt as though he were gagging, and fainted. It is not known exactly how long the patient was unconscious but when he recovered he felt well for the remainder of the night. He reported on sick call the following morning and no abnormalities were found on physical examination, but an electrocardiogram (EKG) taken at that time showed a left bundle branch block (fig. 1). The soldier was called from work to the hospital on 14 April 1950. He was feeling well and had no complaints when he entered the hospital.

On physical examination the pulse was 60 and a slurring of the first heart sound was heard at the apex. The patient's course in the hospital was uneventful. There was no fever. The pulse varied from 45 to 70 beats per minute and was regular. The patient was kept in bed for 4 weeks with no other form of therapy, and he remained asymptomatic while in the hospital. Serial EKG's showed a persistent left bundle branch block with changes suggestive of myocardial infarction (fig. 2). He was transferred to Madigan Army Hospital, Tacoma, Wash., in excellent condition 5 weeks after admission.

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[1] U. S. Air Force Hospital, Fort Richardson, Alaska.

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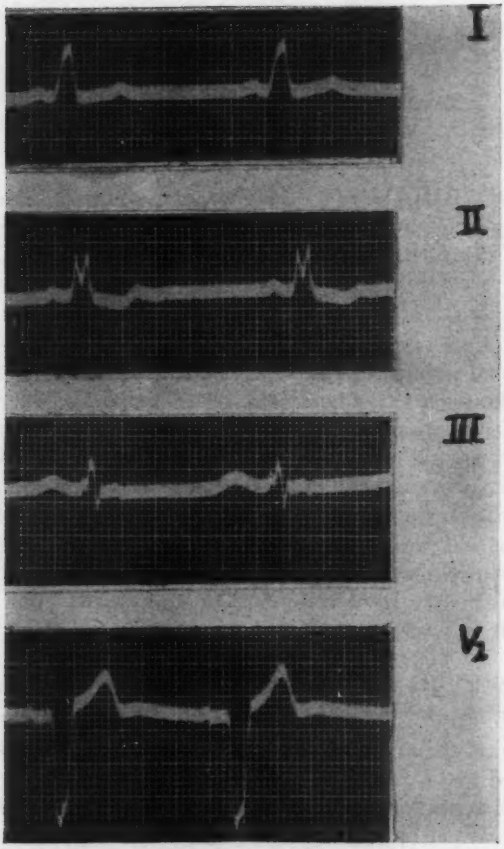

Figure 1.—Case 1. EKG taken 17 April 1950. Note prolongation of QRS complex and slurring and notching. Also elevation of ST and chest lead V_1.

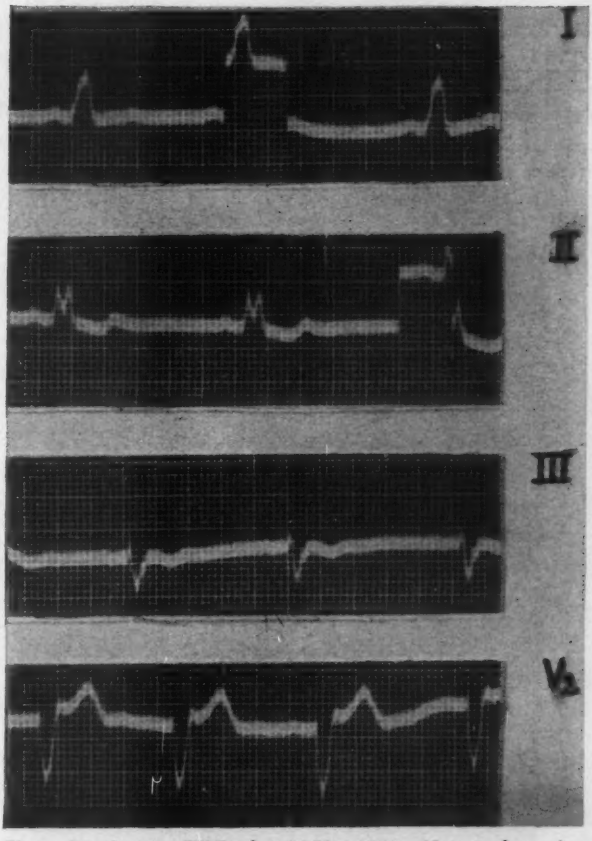

Figure 2.—Case 1. EKG taken 15 May 1950. Note prolongation of QRS complexes with slurring and notching, and elevation of ST segment in chest lead V_2.

Case 2.—A 20-year-old airman, was seized with a sudden severe substernal pain and feeling of tightness while on duty at Cape Air Force Base, Aleutian Islands, 13 April 1950. These symptoms were accompanied by dyspnea, weakness, and sweating. For 2 years prior to this attack, he had had occasional episodes of severe aching substernally, associated with exertion, relieved by rest. There was no history of rheumatic fever, joint or tendon pain, or prior knowledge of cardiac disease. Physical examination revealed a well-developed man who appeared to be acutely ill. The skin was warm and dry. There was no rash. There was dullness to percussion over the left side of the chest posteriorly from the seventh rib to the base, and the breath sounds, tactile, and vocal fremitus were decreased over this area. There were a few scattered rales in the left side of the chest. The pulse was 90. The blood pressure was 120/80. There were no mur-

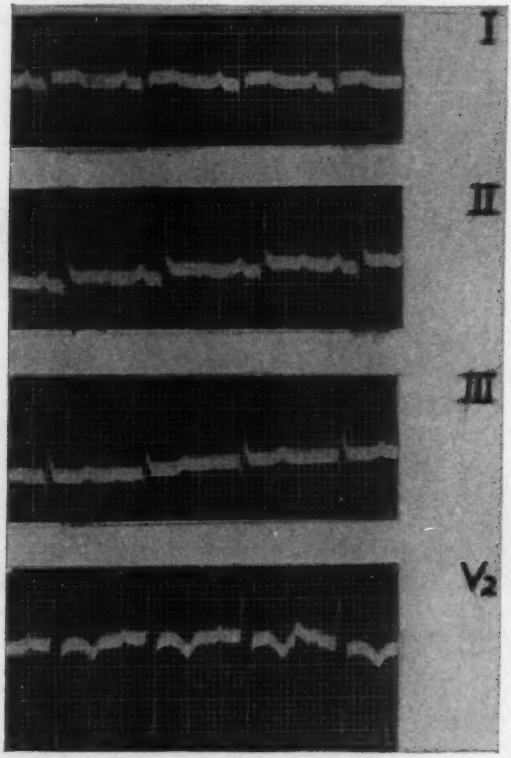

Figure 3.—Case 2. EKG taken 17 April 1950. Note elevation of ST segments and inversion of T in chest lead V_2.

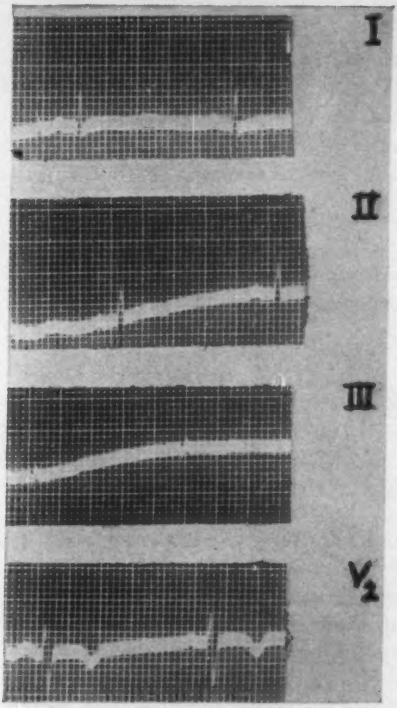

Figure 4.—Case 2. EKG taken 1 May 1950. Note ST segments flat. T_1 in chest leads still inverted.

murs. A definite pericardial friction rub was heard at the left fourth interspace beneath the sternum. The oral temperature was 101° F.

The patient was transferred to this hospital with a diagnosis of pericarditis, secondary to old myocardial infarction or rheumatic fever. The physical findings were as described. The urine contained from 10 to 15 granular casts per high-power field. The hemoglobin was 12.4 grams per 100 cc. The sedimentation rate was 36. Blood culture was negative. An EKG (fig. 3) taken on 17 April 1950 revealed changes consistent with anterior myocardial infarction. A roentgenogram of the chest was negative except for slight cardiac enlargement. In the hospital the patient gradually and progressively improved. The sedimentation rate gradually decreased to 16 within 4 weeks. Serial EKG's showed progressive improvement (fig. 4). The treatment consisted of bed rest, sedation, and supportive measures. Four weeks after admission the patient was transferred to Madigan Army Hospital, Tacoma, Wash., in excellent condition.

DISCUSSION

Very few reports of cases of myocardial infarction in young persons have appeared in the literature. The youngest patient, an 18-year-old male, was reported on by Jameson and Hauser.[2] Zinn and Cosby[3] reported 679 cases of routine postmortem examinations with myocardial infarction analyzed statistically with the results as shown in table 1.

TABLE 1.—*Autopsies in 679 cases of myocardial infarction*[1]

Age	Male	Female	Age	Male	Female
20 to 24	1	0	60 to 64	59	29
25 to 29	0	1	65 to 69	84	43
30 to 34	2	1	70 to 74	89	53
35 to 39	6	1	75 to 79	82	37
40 to 44	11	1	80 to 84	31	17
45 to 49	12	6	85 to 89	8	4
50 to 54	29	10	90 to 94	1	1
55 to 59	45	15			

[1] ZINN, W. J. and COSBY, R. S.: Myocardial infarction; statistical analysis of 679 autopsy-proven cases. Am. J. Med. 8: 169–176, Feb. 1950.

Fagin and Chapnick[4] reported 100 ambulatory patients with electrocardiographic findings of myocardial infarction. They classified these patients as (1) asymptomatic, (2) typical, and (3) atypical. In the first group there were no symptoms referable to the heart; in the second group the patients had histories of acute episodes compatible with the clinical findings of myocardial infarction and followed by disability of varying duration; and in the third group the patients had symptoms of cardiac insufficiency of varying degrees of severity but no history of an acute attack of chest pain of sufficient severity to warrant recognition by the patient, or his physician, as a heart attack. The age incidence in these groups was as shown in table 2.

TABLE 2.—*Age distribution in 100 patients with myocardial infarction*[1]

Age (years)	Asymptomatic	Typical	Atypical	Total
20 to 29			1	1
30 to 39		3	1	4
40 to 49	2	12	7	21
50 to 59	2	20	22	44
60 to 69	3	15	10	28
70 to 79		2		2

[1] FAGIN, I. D., and CHAPNICK, H. A.: Clinical patterns of myocardial infarction in ambulant patients. Ann. Int. Med. 32: 243–256, Feb. 1950.

[2] JAMESON, S. C., and HAUSER, G. H.: Angina pectoris in youth of 18. J. A. M. A. 85: 1398–1399, Oct. 31, 1925.

[3] ZINN, W. J., and COSBY, R. S.: Myocardial infarction; statistical analysis of 679 autopsy-proven cases. Am. J. Med. 8: 169–176, Feb. 1950.

[4] FAGIN, I. D. and CHAPNICK, H. A.: Clinical patterns of myocardinal infarction in ambulant patients. Ann. Int. Med. 32: 243–256, Feb. 1950.

In the two cases here reported it was considered that myocardial infarction could be diagnosed from the EKG's. In case 1 there was left bundle branch block. During this patient's hospitalization the series of EKG's from 13 April to 15 May showed no change. This is usually the case in bundle branch block; that is, the changes are permanent. These patients often live normal lives without limitation of activity. In case 2, however, one can see improvement in the tracings over a 1-month period, as is so often the case in anterior or posterior myocardial infarctions. This man, too, may live a normal life. In young persons myocardial infarctions are most frequently nonfatal because the recuperating potentialities of the youthful myocardium are excellent.

CONCLUSIONS

It is strongly urged that all physicians who see young patients carefully evaluate pain in the left side of the chest. This is a problem testing to the utmost the astuteness of the general practitioner and internist.

Pancytopenia Following the Use of Mesantoin and Thiantoin

Report of a Fatal Case

HARRY A. WEISS, *Lieutenant Commander, MC, U. S. N.*[1]
WILLIAM D. HAWLEY, *Lieutenant, junior grade, MC, U. S. N. R.*[1]

THIS case report is submitted as an illustration of the lethal effects of mesantoin and thiantoin. Best and Paul[2] have recently summarized the reports of toxicity caused by anticonvulsive agents. These reports appear to be increasing proportionately to the increased use of the drugs. The same authors reported the first case of pancytopenia following the administration of thiantoin. Thiantoin is 5,5–phenyl–thienyl–hydantoin and mesantoin is 3–methyl–5–phenyl–5–ethyl–hydantoin.

CASE REPORT

A 28-year-old white woman was first admitted to the hospital on 8 August 1949 because of attacks of fainting and pains in her head of 7 years' duration, underweight, and marked fatigue. The examination and supplemental studies were essentially negative. She was discharged 12 August with a diagnosis of psychogenic asthenic reaction. On 29 August she consulted a physician because of similar complaints to which had been added generalized convulsions. Another physician had witnessed one of the convulsive seizures. The examination was again negative. Laboratory findings, including a complete blood count, were within the normal limits. On 8 September treatment with 0.1 gram of mesantoin twice daily was begun. On 19 September the dose of mesantoin was reduced to 50 mg. twice daily. Treatment with 0.12 gram of thiantoin daily was begun on 18 November. A summary of all medication given is shown in table 1.

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[1] U. S. Naval Hospital, San Diego, Calif.

[2] BEST, W. R., and PAUL, J. T.: Severe hypoplastic anemia following anticonvulsant medication; review of literature and report of case. Am. J. Med. 8: 124–130, Jan. 1950.

TABLE 1.—*Summary of medication given*

Drug	Dose (grams)	Date started	Date discontinued
Mesantoin	0.10 b. i. d.	8- 9-49	9-19-49
Mesantoin	0.05 b. i. d.	9-19-49	1-24-50
Mebaral	0.06 t. i. d.	9-18-49	9-19-49
Desiccated thyroid	Unknown	9-19-49	1-24-50
Thiantoin	0.12 daily	11-18-49	1-24-50
Penicillin	Unknown	1-10-50	1-24-50

She visited her physician on 23 December, and reported that her health was the best it had been in years and that she had had only one petit mal seizure. Physical examination was again negative. She next consulted a physician on 10 January 1950 because her gums were sore and bled easily. She was treated with penicillin intramuscularly but the condition persisted. She consulted a dentist on 20 January and was given penicillin ointment for local application.

She was readmitted to the hospital on 24 January complaining of severe weakness and profuse vaginal bleeding of 4 days' duration. Examination revealed a thin, pale, lethargic, acutely ill woman. She responded to questioning, although her responses were hazy. There were multiple purpuric areas on the skin of the arms and legs. These had been present for 2 weeks. The alveolar margins were hyperemic and had shallow ulcerations covered with a gray membrane. They bled easily on contact. There were several lesions of similar appearance on the posterior pharyngeal wall. The examination was otherwise negative. The erythrocyte count was 1,200,000 with 5.5 grams of hemoglobin. The packed cell volume was 17 percent, and the leukocyte count was 1,100, with lymphocytes 100 percent. No platelets were seen. Urinalysis was negative. The icterus index was 5; the serum bilirubin was 0.35 mg. per 100 ml. The thymol turbidity was 1 unit and the cephalin cholesterol flocculation test was one plus in 48 hours.

Treatment consisted of supportive measures, antibiotics, and the substances thought to have hematopoietic properties. Therapy and hematologic studies are summarized in table 2.

Eighteen hours after admission, following transfusions of whole blood, the patient seemed improved clinically and was alert and responsive, although still pale. She complained of a headache. Twenty-four hours after admission she felt dizzy and had a few mild generalized tremors. She was nauseated and vomited shortly after these. She was very restless that night. Sternal marrow obtained on the third hospital day showed (after 2,000 cc. of whole blood had been given), 1 myeloblast, 2 myelocytes, 2 eosinophils, 5 erythroblasts, and 2 normoblasts in 400 cells counted. The remainder of the cells were lymphocytes. That day the patient developed fever and her

TABLE 2.—*Summary of blood findings and therapy during second hospitalization, January 1950*

Day of month	Hemoglobin (grams per 100 cc.)	Red blood cells (million)	White blood cells	Polymorphonuclear cells (percent)	Platelets (thousand)	Sodium amytal (grams)	Whole blood (cc.)	Pentnucleotide (cc.)	Crude liver extract (cc.)	Vitamin B_{12} (micrograms)	Penicillin (1,000 units)	Aureomycin (grams)
24	5.5	1.00	1,100	0	None	0.2	1,000	40	20		400	0.25
25	8.0	2.60	850	0	None		1,000	40	20		400	0.25
26			350									
27		3.25	800	0	Normal		1,500	40	20		400	0.25
28	13.0	4.50	550	0	110	0.4		40	20	15	400	0.25
29	13.0	3.80	1,100	0	260	0.2	1,000	40	20		400	0.25
30		4.10	300	0	80	0.6	1,000	40	20		400	0.25
31	14.0	4.70	400	4		0.4	500	40	20		400	0.25
	15.0	5.10	350	0	220		1,000	40	20		400	0.25

temperature rose to 105° F. The fever was accompanied by severe diarrhea and the stools contained bright red blood. The fever continued through the next 2 days. On the sixth hospital day she developed frequency of urination and gross hematuria. She continued to have the same symptoms and findings, gradually becoming weaker. On the seventh hospital day she began having shallow, rapid respirations, cyanosis, and a cough productive of mucopurulent sputum. The lung bases became definitely congested. Within the next 6 hours she became severely jaundiced and lapsed into coma. Death occurred 7 days and 10 hours after admission.

The autopsy showed generalized purpura and icterus. Many of the purpuric lesions were necrotic. There was bilateral pneumonitis and a left pleural effusion. There was uterine hemorrhage. The bone marrow was hypoplastic throughout.

SUMMARY

This patient died following the use of mesantoin and thiantoin. These drugs are not without dangerous hematologic complications. Blood studies should be made at least every 2 weeks during their administration.

Hypogonadism

Puberal Seminiferous Tubule Atrophy

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ROBERT A. KALLSEN, *M. D.*[2]

UNTIL recently, the presence of testicular inadequacy in young men was generally recognized only in those who had early androgen failure with body disproportion characteristic of eunuchoidism. That hypogonadism may occur in the young adult with an apparently normal body type was reemphasized in the report by Klinefelter, Reifenstein, and Albright [3] on the occurrence of gynecomastia, aspermatogenesis with androgen secretion by the interstitial cells of Leydig present, and increased urinary follicle-stimulating hormone.

Heller et al.[4-7] expanded our knowledge further when they classified their patients according to the time of onset of testicular failure, the resulting body disproportions, the urinary excretion of gonadotropin, and the type of pathologic changes involved. In their classification, the condition in hypogonadal patients with or without gynecomastia, but with seminiferous tubule atrophy occurring before or during puberty, was termed "puberal seminiferous tubule atrophy." These patients were subdivided into noneunuchoid, moderately eunuchoid,

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[3] KLINEFELTER, H. F., JR.; REIFENSTEIN, E. C., JR.; and ALBRIGHT, F.: Syndrome characterized by gynecomastia, aspermatogenesis without A-leydigism, and increased excretion of follicle-stimulating hormone. J. Clin. Endocrinol. 2: 615–627, Nov. 1942.

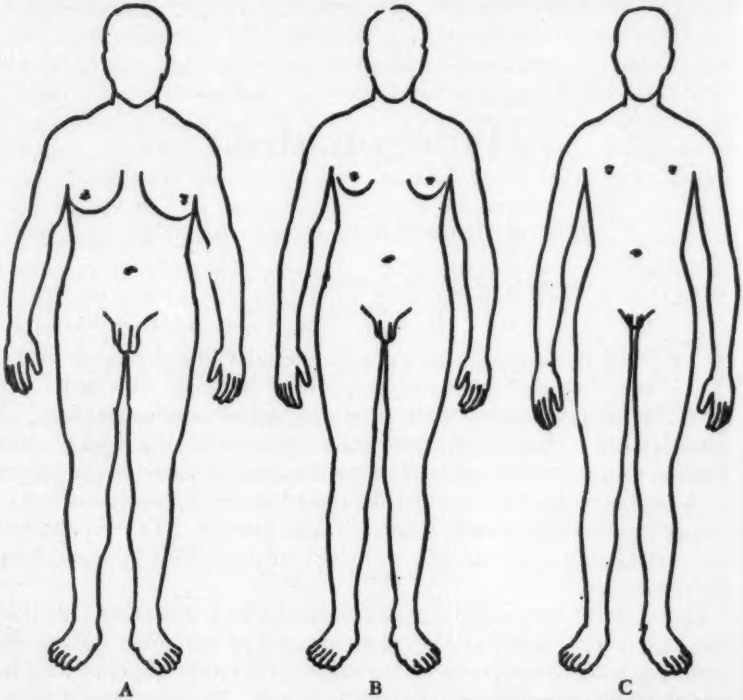
[4] HELLER, C. G.; NELSON, W. O.; and ROTH, A. A.: Functional prepuberal castration in males. J. Clin. Endocrinol. 3: 573–588, Nov. 1943.

[5] NELSON, W. O., and HELLER, C. G.: Hyalinization of seminiferous tubules associated with normal or failing Leydig-cell function; microscopic picture in testis and associated changes in breast. J. Clin. Endocrinol. 5: 13–26, Jan. 1945.

[6] HELLER, C. G., and MADDOCK, W. O.: Clinical uses of testosterone in male. Vitamins & Hormones 5: 393–432, 1947.

[7] HELLER, C. G., and NELSON, W. O.: Classification of male hypogonadism and discussion of pathologic physiology, diagnosis, and treatment. J. Clin. Endocrinol. 8: 345–366, May 1948.

and eunuchoid types, according to the size of the gonads and the body characteristics.[8] In their series the most marked gynecomastia oc-



—Courtesy of the Journal of Clinical Endocrinology

Figure 1.—Body types, breast, and genital development in hypogonadism. (A) *Noneunuchoid.* (B) *Moderate eunuchoid.* (C) *Eunuchoid.*[8]

curred in patients with the least testicular atrophy and the most definite male characteristics (noneunuchoid). The relationship of the body characteristics and the degree of gynecomastia are shown in figure 1.

Although gynecomastia is not a necessary part of hypogonadism, the description by Klinefelter et al. is useful and emphasizes several of the diagnostic points which should lead the clinician to suspect hypogonadism. These are small insensitive testes without sperm production, an increased excretion of urinary gonadotropin, gynecomastia, and certain typical body disproportions.

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[8] HELLER, C. G., and NELSON, W. O.: Hyalinization of seminiferous tubules associated with normal or failing Leydig-cell function; discussion of relationship to eunuchoidism, gynecomastia, elevated gonadotrophins, depressed 17-ketosteroids and estrogens. J. Clin. Endocrinol. *5*: 1–12, Jan. 1945.

These patients are usually unaware of the significance of their endocrine disorder and have a natural reluctance to draw attention to their physical abnormalities. Usually they do not seek treatment for their unusual characteristics. Their endocrine dysfunction is often overlooked or considered not to warrant treatment. Because diagnosis is a relatively simple matter, and treatment with testosterone is of considerable benefit, it is believed that a discussion of this condition and a review of three typical cases may serve a useful purpose.

The condition is believed to begin during or before adolescence with atrophy of the tubules and disappearance of the germinal epithelium and Sertoli cells.[3] The cause of it is obscure. Most patients give a history of childhood or pubertal infectious process such as mumps with or without orchitis.

In spite of the absence of tubular elements, the continued androgen production by the Leydig cells results in normal development of the

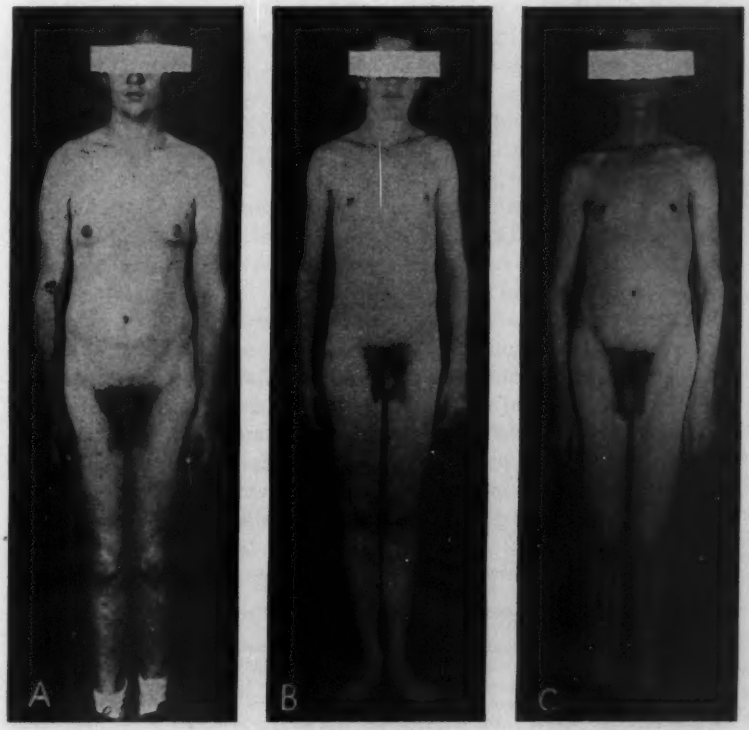

Figure 2.—(A) *Patient 1, noneunuchoid.* (B) *Patient 2, moderate eunuchoid.* (C) *Patient 3, eunuchoid.*

penis, genital tract, prostate, and pubic hair. Testicular biopsy (usually not necessary for diagnosis) reveals a dense stroma of Leydig cells with scattered atrophic hyalinized tubules and no germinal elements. Associated with these findings are those common to eunuchoidism (noted in our three cases), namely, a low basal metabolic rate with a normal blood cholesterol, a low fasting blood sugar with a flat glucose tolerance curve, and a low resting blood pressure and pulse. Patients with these findings are often treated with thyroid extract. This does not in any way alleviate the condition.

An important characteristic of this syndrome is a definite personality deviation.[7] The person may be immature, nervous, unstable, and generally inadequate. These psychologic manifestations are benefited by androgen therapy.

In our three cases the complaints presented by the patient on admission were: a sprained ankle (case 1), painful left gynecomastia with bilateral palpable gynecomastia (case 2), and extreme nervousness with neurotic trends (case 3). All three of our patients (fig. 2) had a penis of normal size, but had definitely atrophic testicles. All had sparse or absent beard and no chest hair. Gynecomastia of some degree was present in each patient. Our patient with the highest androgen excretion and the most male body type, also had the most gynecomastia. Minimal gynecomastia occurred in the eunuchoid patient. This supports the observations of Heller and Nelson as shown in figure 1. Abnormally long extremities were present, but the general body configuration was that of the adult male. The distance from the pubis to the heel exceeded the sitting height in each patient, and armspread exceeded the height in two.

All three patients had increased urinary gonadotropins as measured by the increase in weight of rat ovary and mouse uterus after injection of a concentrated urine specimen.[9] Testicular biopsy in each case revealed atrophic hyalinized tubules with a dense stroma of Leydig cells, as shown in figure 3. No spermatozoa were present in the semen. Ejaculation produced less than 2 cc. of semen in the eunuchoid patient, and about 4 cc. of semen in the two patients with the more masculine characteristics. The examination of the semen for spermatozoa is

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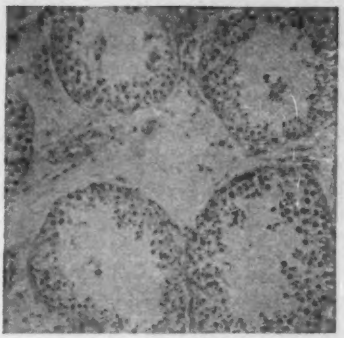
[9] Twenty-four-hour urine specimens were precipitated with alcohol, washed with alcohol and ether, and dialyzed according to Heller and Chandler.[10] The purified precipitate was assayed in 22-day-old rats and mice with ovarian weight, uterine weight, and vaginal opening as the end-points [10 11 12].

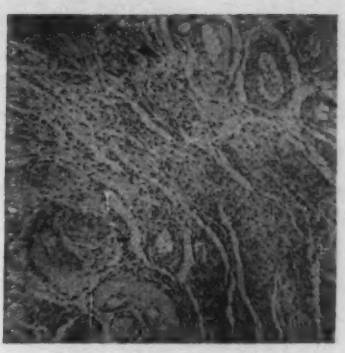
[10] HELLER, C. G., and CHANDLER, R. E.: Gonadotropic hormone; modification of alcohol-precipitation assay method. J. Clin. Endocrinol. 2: 252–253, Apr. 1942.

[11] FEVOLD, H. L.: Extraction and standardization of pituitary follicle-stimulating and luteinizing hormones. Endocrinology 24: 435–446, Apr. 1939.

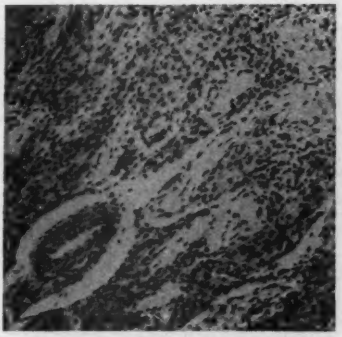
[12] HELLER, C. G.; LAUSON, H.; and SEVRINGHAUS, E. L.: Immature rat uterus as assay end-point for gonadotropic substances. Am. J. Physiol. 121: 364–378, Feb. 1938.



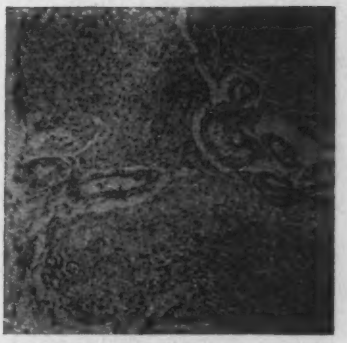
Normal



Patient 1



Patient 2



Patient 3

Figure 3.—Biopsy sections contrasting the normal with those showing atrophic hyalinized tubules in a dense stroma of Leydig cells.

rapid and simple and should be done in any patient suspected of having hypogonadism.

A history of mumps without orchitis was obtained in case 1; mumps with orchitis in case 2; and no mumps, trauma, or infectious process involving the testicles in case 3. All three patients had normal libido, erections, and ejaculations, and had had successful sexual intercourse. All three complained of easy fatigability, extreme nervousness, and inability to arouse easily in the morning.

Psychiatric evaluation on admission revealed that in case 1, the patient was dull, apathetic, and tremulous. He had been dismissed from school and from his previous job for disciplinary reasons. He was classified as having a personality defect with immaturity and emotional instability. The patient in case 2 had also been dismissed from school, and was classified as having an inadequate, immature, psycho-

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pathic personality. The patient in case 3 had left school at the eighth grade because of lack of interest. He had many fears, and during the past year had noted excessive nervousness. He was classified as a schizoid personality. All three were nail biters.

The laboratory findings of significance were the low basal metabolic rate with normal blood cholesterol, low fasting blood sugar and flat glucose tolerance curve, increased urinary gonadotropins, and absence of spermatozoa (table 1).

TABLE 1.—*Laboratory findings*

	Patient 1 (noneunuchoid)	Patient 2 (moderate eunuchoid)	Patient 3 (eunuchoid)
Urinary gonadotropins	Increased (100 M. U.)	Increased (300 M. U.)	Increased (300 M. U.).
Sperm count	0	0	0.
Urinary 17-ketosteroids	10 to 15 mg./24 hr.	6 to 8 mg./24 hr.	6 mg./24 hr.
Testicular biopsy	Atrophic tubules. Dense Leydig cell stroma	Atrophic tubules. Dense Leydig cell stroma	Atrophic tubules. Dense Leydig cell stroma.
Average basal metabolic rate.	−27	−30	−42.
Sleeping pulse	45	55	48.
Average blood cholesterol	140 mg.	155 mg.	163 mg.
Blood sugar:			
Fasting	65 mg.	79 mg.	85 mg.
After 100 grams glucose	68 mg.	81 mg.	81 mg.
Bone age	Normal	Normal	Normal.
Serum inorganic phosphorus.	2.8 mg.	5.2 mg.	6.0 mg.
Circulating lymphocytes:			
Before treatment	1,800/cu. mm.	2,900/cu. mm.	2,400/cu. mm.
During testosterone therapy.	2,500/cu. mm.	3,200/cu. mm.	4,080/cu. mm.

It is of interest that patient 1 was first given a course of desiccated thyroid, 120 mg. daily, which in no way altered his condition or changed his basal metabolic rate. All three patients were eventually treated with testosterone; first parenterally, and then by sublingual or buccal pouch administration. No increased effectiveness was noted with the parenteral administration and the buccal pouch was used because of its simplicity. Little change was noted by laboratory tests, but the patients were subjectively greatly improved. All three remarked on the ease with which they could arise in the morning; they stated that they felt generally more alive and were capable of a much greater work output. Their nervousness decreased, and they were more useful around the ward. Disciplinary infractions were greatly decreased. Psychiatric reevaluation after a month of therapy showed definite general personality improvement in two of the patients, and a decrease in nervousness in the third.

The basal metabolic rate rose to normal levels in patient 2 when 100 mg. of testosterone propionate daily was given, and in patient 1 when 50 mg. daily was given. Although more frequent erections occurred on the higher dosage, in none did these symptoms become distressing. Later when the patients were started on sublingual tes-

tosterone with an initial dosage of 18 mg. daily, all three complained of nearly continual erections. With a reduction of dosage to 12 mg. daily, this ceased. Although this amount did not bring the basal metabolic rate up to normal, nor apparently affect the sleeping pulse to any degree, it achieved the desired effect subjectively. All patients improved remarkably. They were more alert and energetic. They stated that they felt stronger and had more stamina. Regardless of the dosage or route of administration, the blood sugar curves remained flat in the three patients, and the urinary gonadotropins remained above normal.

The differentiation of primary hypogonadism, or direct impairment of testicular function from secondary hypogonadism resulting from pituitary dysfunction, can be established by both clinical and laboratory findings. The pituitary (secondary) hypogonadal patient has a generalized adolescent development; no gynecomastia is present; voice, beard, and pubic hair are of a preadolescent type; the testes and penis are small; the prostate is small or absent; and the bone development is retarded as shown by open epiphyses. In primary hypogonadism which occurs after or during puberty, the sex characteristics are of adult type; there is usually gynecomastia; the voice is normal; the beard may be scant, chest hair is usually absent, but there is normal adult pubic hair; the testes are small and atrophic, but the penis is of normal size; the prostate is within normal limits; and the bone development is not retarded.

In primary hypogonadism (in the castrate, or in testicular failure) an increased urinary gonadotropin is present. In secondary hypogonadism (the pituitary insufficiency type of hypogonadism) there is a decreased urinary gonadotropin. Testicular biopsy in these two types will also show typical differences; in primary testicular failure there is absence of all tubular cellular elements (germinal epithelium and Sertoli cells), whereas in the secondary or pituitary failure, immature germinal elements are present in the testes.

CASE REPORTS

Case 1.—A 25-year-old unmarried white man was seen in December 1948 because of a sprained ankle. In the course of a routine physical examination it was discovered that the patient had bilateral gynecomastia and atrophic testicles. He had mumps without orchitis at the age of 9. At 11 years of age bilateral tenderness and swelling of the breasts were first noted; this tenderness persisted until the age of 17. Pubic hair appeared at the usual age. Libido was normal and there had been no difficulty in obtaining erection or accomplishing ejaculation. He had sexual intercourse first at the age of 15. He

began to shave at 23 years of age and now shaves once a week. He had noticed that in the last 3 or 4 years he tired and became cold easily, and required more blankets than other people. He perspired freely; no dryness of the skin had been noted. For the past 4 years he has had considerable tremor of the hands.

There was no family history of endocrinopathy.

Examination on admission revealed a well-developed white man. The arm span was not significantly greater than the height. The beard was sparse and pubic hair was abundant, and although there was no chest hair the general distribution was within normal male limits. The fingernails were bitten to the quick. The breasts were enlarged, nontender, and female in type. The penis, scrotum, and prostate were well developed. The testes were small and about 1½ cm. in diameter. There was no tenderness on pressure of the right testis and only slight tenderness on the left.

Roentgenograms showed normal bone age. Urinalysis and complete blood count were normal.

The patient was first given a 6-week course of desiccated thyroid, 120 mg. daily, without any effect on his basal metabolic rate or his symptoms except to increase his nervousness. Testosterone propionate was then prescribed and he noted almost immediate decrease in nervousness, felt more energetic, his stamina increased, and he began to help with the ward chores without urging. Although he previously refused to be aroused in the morning in spite of disciplinary action, he now arose early of his own accord. With this personality change there was a moderate increase in basal metabolic rate.

Case 2.—A 19-year-old unmarried white man was first seen in February 1949 complaining of pain and tenderness of the left breast which he first experienced 2 years previously. Intermittent tenderness and occasional episodes of swelling had been experienced during this period. All symptoms had been confined to the left breast. The onset of puberty and the development of pubic hair and other secondary sex characteristics occurred at about the usual age. Libido was normal and he had no difficulty in obtaining an erection or accomplishing ejaculation. Sexual intercourse first occurred at the age of 15. There was no history of heat or cold intolerance. He had mumps and orchitis at the age of 5. Family history revealed no evidence of endocrinopathy.

Physical examination revealed a well-developed white man who did not appear ill. The arm span was 3 inches more than the height. Facial hair was absent. Axillary and pubic hair was sparse but within normal limits. There was no chest hair. The fingernails

were bitten to the quick. The breasts were slightly enlarged with palpable glandular tissue; the left breast was moderately tender. The penis, scrotum, and prostate were well developed. The right testis was just barely palpable; the left was approximately the size of a pea and nontender. Roentgenograms revealed normal bone age.

Routine laboratory examinations were normal.

During therapy with as much as 100 mg. testosterone propionate daily, the main change noted was an increase in the basal metabolic rate. This patient, whose personality deviation was more definite than in case 1, did not respond entirely to therapy but did become more stable and was easier to manage.

Case 3.—A 20-year-old unmarried white man was first seen in April 1949 complaining of spells of "blacking out." He was seen on the neuropsychiatric service and a diagnosis of a severe anxiety neurosis was made. He was referred to the endocrine clinic because of small testes. He had always had small testes and had been weak and of slight build. He had normal libido, erections, and ejaculations and first had intercourse at the age of 18. He shaved twice a week. He had never had mammary swelling or soreness. He always has had some intolerance to cold, easy fatigability, and difficulty in being aroused in the morning. Past history revealed no evidence of mumps or any testicular trauma.

Examination on admission revealed a poorly developed asthenic white man appearing extremely nervous and apprehensive. The beard was practically absent, and the distribution of pubic hair tended toward the female type. Axillary hair was sparse. There was no chest hair. A mild gynecomastia was present bilaterally. Both testes were atrophic, about 1 cm. in diameter, and only slightly tender to pressure. The penis and scrotum were well developed. The prostate was small but within normal limits. No prostatic secretion was present.

Roentgenograms revealed normal bone age.

The response to therapy was dramatic and a very definite personality disturbance was alleviated.

SUMMARY

Three cases of a syndrome characterized by aspermatogenesis without the absence of androgen secretion of the interstitial cells of Leydig, and increased urinary gonadotropins have been presented. Because of the relative lack of gynecomastia in the eunuchoid patient and the extreme gynecomastia in the patient with male body structure, it is believed that the syndrome described by Klinefelter is not specific,

and that these patients represent an incomplete eunuchoid state with body characteristics dependent on the degree and time of onset of failure. Testosterone therapy produces favorable results. In these three patients, testosterone administered sublingually or between the cheek and teeth produced clinical results equal to those from parenterally administered testosterone propionate. This similarity in response has not been observed in all patients with hypogonadism.

Study of Five Hundred Autopsies in Cases of Tuberculosis

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HENRY M. GREENLEAF, *Lieutenant Colonel, MC, U. S. A.*[1]

FROM 1944 through 1947, 9,388 patients with tuberculosis were admitted to this hospital. In the same period, there were 342 deaths from tuberculosis (all types). We were impressed with the fact that our clinical and autopsy material was not consistently demonstrating the extrapulmonary complications that we had been led to expect, and that was one reason for undertaking this study. The same senior pathologist supervised the autopsies throughout the period covered by this report. The material consists of 500 consecutive autopsy reports of male patients who died with a primary diagnosis of pulmonary tuberculosis confirmed by the pathologist. No patients who died of other causes or in whom tuberculosis was reported as an incidental finding were included. Patients in whom the primary cause of death was nonpulmonary tuberculosis are included if pulmonary tuberculosis was also present.

Clinical charts were used only for recording the age of the patient at the time of death and for determining the duration of the disease from the first symptoms referable to tuberculosis to the time of death. Although this period is difficult to fix, and depends on the skill and conscientiousness of the many clinicians concerned, it is believed that the accuracy in recording the onset in these patients was as good or better than average. Practically all patients had their onset while in the Federal service, a fact that could be accurately checked by the induction chest roentgenogram in most cases. We believe that the rigors of active duty and the availability of medical observation and laboratory facilities made early detection and diagnosis possible in most of the patients.

The limitations inherent in such a study lay in the fact that (*a*) the patients were all men 17 years or more of age; (*b*) the mission of this

[1] Fitzsimons Army Hospital, Denver, Colo.

hospital is primarily that of a clearing house for diagnosis, initiating treatment, providing surgical treatment where indicated, and then arranging for sanatorium or home care elsewhere;[2] (*c*) the changing staff of assistant pathologists altered the reported autopsy findings to some extent, despite the supervision by the same chief pathologist; and (*d*) from 1939 to 1942 inclusive the cranium was usually opened for examination only when intracranial tuberculosis was suspected clinically.[3]

MATERIAL

Race.—Of the 500 cases studied, 369 patients were white, 123 were Negroes, and 8 were Indians.

Age.—Although the average age for the entire series was 35.1 years only 99 were in the 30- to 39-years age group, 224 were below 30 years, and 177 were above 40. The oldest patient was 77 and the youngest was 18 years old. Further distribution is given in table 1.

TABLE 1.—*Distribution by age groups*

Age in years	Number in 1939–43	Number in 1944–47	Total
Under 20	6	6	12
20 to 29	90	122	212
30 to 39	49	50	99
40 to 49	95	13	108
Over 50	50	19	69
Total	290	210	500

Extent and type of pulmonary involvement.—Eight cases were moderately advanced and 492 were far advanced. A mixed pulmonary lesion was found in 483; miliary involvement was found in 11, nodose involvement in 4, and pneumonic involvement in 2. Cavitation was found in 433.

Duration of infection is shown in table 2. The longest duration in a white patient was 26 years and in a Negro patient, 28 years; the shortest was 1 month for both white and Negro patients. The average duration for white patients was 4 years and 6 months but for Negroes was only 1 year and 7 months. In this computation we excluded two atypical cases, the white patient with a duration of 26 years and the

[2] A large percent of the patients dying at this hospital were acutely ill young adults and a smaller number were veterans of World War I whose disease was chronic and who came to this hospital to die. Immediately after the cessation of hostilities many prisoners of war with advanced disease secondary to terrific privation who were admitted died. An increase in the number of Negro patients resulting from the increased percent of Negro troops in the services in World War II accounted for another large group of terminal cases.

[3] The incidence of tuberculoma, miliary or meningitic involvement within the skull did not, however, show appreciable change in the period from 1943 to 1947 when the brain was examined in every instance.

Negro with a duration of 28 years. Of 355 patients who had their illness less than 3 years, 220 were under 30 years of age. Forty-six died in less than 6 months after the onset of symptoms. Of the 429 patients who had their illness less than 10 years, 80 were over 40 years of age.

TABLE 2.—*Duration of infection*

	Number
Less than 1 year	209
1 to 2 years	146
3 to 4 years	33
5 to 9 years	41
Over 10 years	71
Total number	500

Longest duration:	
White	26 years.
Negro	28 years.
Shortest duration:	
White	1 month.
Negro	1 month.
Average duration for the group	4 years and 6 months.
Average duration for Negroes	1 year and 7 months.

FINDINGS

The complications found in this series are shown in table 3. Pleurisy, pleural effusion, pleural adhesions, bronchopleural fistulas and bronchocutaneous fistulas were not recorded. Generalized or specific organ wasting or atrophy was not considered a complication. Simple atrophy of endocrine glands was of interest but was not recorded because of the difficulty of establishing an adequate line of demarcation between the normal and the abnormal. Paranasal sinus and tonsillar involvement were not recorded, because sinuses were not examined and tonsils, if present, were not sectioned routinely.

Pulmonary hemorrhage was listed as a terminal complication only when it was believed to be the immediate cause of death either by exsanguination or suffocation; the finding of gross blood is not positive proof of death by hemorrhage. Such hemorrhage was found in 54 patients; only 2 of these showed no cavitation. Thirteen of the 54 were Negroes. The average age at death associated with hemorrhage was 35.9 years, and the average duration of the disease at death was 4 years and 9 months. The longest duration of the disease at time of hemorrhage and death was 26 years and the shortest was 2 months. The oldest patient at time of hemorrhage and death was 65 and the youngest was 19 years of age. Because Negroes made up 24 percent of those who died with hemorrhage and 24 percent of the whole series,

this complication did not appear more frequently among the Negroes in our series than among the white patients. The average duration of the disease at time of death, associated with hemorrhage, was about the same as the duration for the group as a whole.

TABLE 3.—*Complications found at autopsy*

Involvement	*Number*
Gastrointestinal tract	250
Liver	222
Spleen	208
Lymph glands (other than hilar)	208
Kidney	146
Larynx	132
Clubbing of fingers	110
Genital organs	79
Adrenal glands	70
Peritoneum	69
Trachea	68
Central nervous system	51
Tuberculoma	15
Meningitis	34
Other	2
Amyloidosis	36
Thyroid	25
Bone	21
Pericardium	17
Pancreas	9
Myocardium	9
Ear	8
Skin (other than around fistulas)	4

Empyema was considered to be present only in those cases in which frank pus was found in the pleural cavity at autopsy. One hundred and four cases showed this complication. The longest duration of disease in this group was 21 years, and the shortest, 2 months (average 3 years and 8 months). Forty-five of these had not had collapse therapy; 38 had received pneumoperitoneum or pneumothorax; and 21 had had thoracoplasty.

Cor pulmonale was a terminal or contributory cause of death in many patients. Although it was difficult to establish criteria for such a diagnosis from the autopsy reports, dilatation of the right side of the heart or hypertrophy was present in about 205 patients.

Metastatic or extrapulmonary tuberculous involvement.—No extrapulmonary involvement of any kind, including clubbing of the fingers and amyloidosis, was found in 54 patients. In this group the average age was higher than that of the group as a whole. The oldest was 67 and the youngest 22 years old. Sixteen were below 40 and 24 were above 50 years of age. The incidence of extrapulmonary tuberculous

involvement is shown in table 3. In the 146 patients with renal involvement, 115 had involvement of both kidneys.

CORRELATIONS

Correlation of the more prevalent complications with the duration of the disease is shown in table 4. An appreciable number (22 percent) of complications was present in that portion of the group with a duration of less than 1 year who died in less than 6 months. Almost all of the patients who died within the first 3 years after onset of the disease were within the group 20 to 35 years of age, and those who had had symptoms for 10 years or more before death were over 50 years of age. The percentage of gastrointestinal, liver, spleen, and lymph gland involvement in the cases of less than 3 years duration was high. Ear involvement was significantly greater in the cases of short duration. Clubbing of the fingers had little relation to the duration of the disease. Amyloidosis was found in the cases of longer duration. Correlation of the extrapulmonary complications with chronologic age, at the time of death, is shown in table 5.

TABLE 4.—*Correlation of complications with disease duration*

Duration in years	Less than 1	1 to 2	3 to 4	5 to 9	Over 10	Total
	PERCENT					
COMPLICATIONS						
Gastrointestinal tract	58.2	51.3	33.3	40.4	37.5	50.0
Liver	55.8	41.6	36.1	23.8	27.7	44.4
Spleen	53.3	40.2	33.3	26.1	23.6	41.6
Lymph glands (other than hilar)	50.4	36.1	33.3	38.0	33.3	41.6
Kidney	35.4	21.5	25.0	26.1	30.5	29.2
Larynx	31.5	27.0	38.8	11.9	12.5	26.4
Clubbing of fingers	21.8	25.0	19.4	16.6	20.8	22.0
Genital organs	16.9	20.1	22.2	14.2	15.2	15.8
Adrenal glands	15.5	13.1	5.5	14.2	15.2	14.0
Peritoneum	17.9	15.9	11.1	4.7	4.1	13.9
Trachea	16.5	13.8	11.1	11.9	6.9	13.8
Central nervous system:						
Tuberculoma	4.3	2.7	2.7	2.3	0.0	3.0
Meningitis	7.7	9.6	8.3	4.7	2.1	6.1
Amyloidosis	3.3	3.4	16.6	16.6	15.2	7.1
Thyroid gland	4.3	5.5	11.1	4.7	2.7	5.0
Bone	5.3	2.7	5.5	4.7	2.7	4.2
Pericardium	4.3	2.0	0.0	2.3	5.5	3.4
Pancreas	1.9	1.3	2.7	0.0	2.7	1.8
Myocardium	1.4	2.0	5.5	0.0	1.3	1.8
Ear	2.9	1.3	0.0	0.0	0.0	1.7

Sixty-seven patients showed no evidence of pulmonary cavitation. Of this group, 13 had no evidence of extrapulmonary involvement. Thus, about the same proportion showed no evidence of extrapulmonary involvement as for the total group of 500 patients. Although the presence or absence of pulmonary cavitation made no significant difference in the incidence of extrapulmonary complications, the nature of the complications was altered as shown in figure 1.

TABLE 5.—*Correlation of complications with age at time of death*

Age in years	Under 20	20 to 29	30 to 39	40 to 49	Over 50	Total
	PERCENT					
COMPLICATION						
Gastrointestinal tract	58.3	62.1	47	41.1	24.1	50.0
Liver	50.0	54.2	44	27.7	32.7	44.4
Spleen	66.6	50.9	42	26.0	31.0	41.6
Lymph glands (other than hilar)	41.6	49.5	39	42.0	13.7	41.6
Kidney	16.6	26.6	38	30.2	22.0	29.2
Larynx	41.6	31.7	24	24.3	10.3	26.4
Clubbing of fingers	25.0	21.9	21	12.6	41.4	22.0
Genital organs	25.0	14.0	20	15.1	13.7	15.8
Adrenal glands	16.6	14.0	13	13.4	15.4	14.0
Peritoneum	16.6	19.6	9	6.4	10.3	13.9
Trachea	8.3	16.8	14	7.5	13.7	13.8
Central nervous system:						
Tuberculoma	8.3	3.7	4	0.0	1.7	3.0
Meningitis	8.3	8.8	7	5.0	6.9	6.1
Amyloidosis	0.0	3.7	8	10.0	13.7	7.1
Thyroid gland	0.0	7.4	3	1.6	6.9	5.0
Bone	8.3	4.6	5	2.5	3.4	4.2
Pericardium	8.3	1.8	4	2.5	5.1	3.4
Pancreas	0.0	3.2	1	1.3	0.0	1.8
Myocardium	0.0	2.3	1	1.3	0.0	1.8
Ear	0.0	2.8	1	0.0	1.7	1.7

Involvement of the liver, spleen, kidneys, pancreas, thyroid, adrenals, genital organs, central nervous system, myocardium, pharynx, and bone is thus shown to be relatively more frequent in those with pulmonary cavitation than in the entire series, while involvement of the gastrointestinal tract, lymph glands, larynx, fingers, and trachea was less frequent, and involvement of the peritoneum, pericardium, skin, and amyloid formation was little altered.

TABLE 6.—*Percent of patients with empyema compared with clubbing of fingers, bone involvement, and amyloidosis*

	Percent of 104 patients with empyema	Percent of 396 patients without empyema	Percent of entire series
Clubbing of fingers	23.0	21.0	22
Bone involvement	4.7	4.0	4
Amyloidosis	14.4	5.3	7

Of 104 patients with empyema, 24 had clubbing of fingers, 15 had amyloidosis, and 5 had bone involvement (table 6). Clubbing of fingers and bone involvement appear to have no relation to empyema, but the incidence of amyloidosis is definitely increased in the presence of this complication. Amyloidosis seems to have almost disappeared as a complication of pulmonary tuberculosis. Although in the first 290 patients who died between 1939 and 1943, 35 had amyloidosis; in the next 210 patients who died between 1944 and 1947 this complication was found in only 1 despite diligent search. The only explanation that appears likely is that the last part of the series contained a large number of young patients with fulminating disease of short duration.

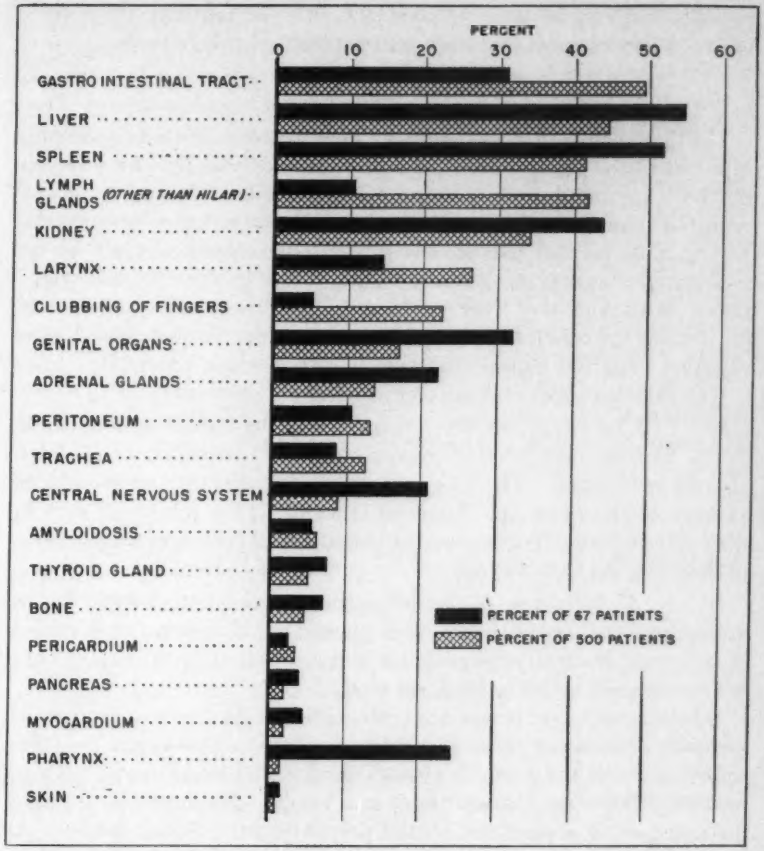

Figure 1.—Incidence of complications in patients without pulmonary cavitation and in entire series.

Of 158 patients with involvement of the larynx and/or trachea, 51 had involvement of both, and 118 had involvement of the gastrointestinal tract as well. There appears to be a significant correlation of these complications, a fact which is probably associated with the presence of positive sputum.

Of 222 patients with liver involvement, 187 had involvement of the spleen, 116 had renal involvement, and 56 had involvement of the adrenals as well, as compared with 14 percent for the entire series. One hundred and nine had involvement of the spleen, liver, and kidneys. Hematogenous spread appeared to have a predilection for the liver, spleen, renal, and adrenal tissue, in that order. The kidneys were diseased in 30, the spleen in 22, and the adrenal glands in 14

patients without hepatic involvement. Of 70 patients with peritonitis, 61 were associated with gastrointestinal tuberculosis.

DISCUSSION

The figures presented are probably weighted with acute fulminating infections in a group whose general resistance was low, as revealed by the large number of cases of short duration. Inasmuch as the reported tissue involvement is based entirely on autopsy findings the fact that 50 percent had lesions in the gastrointestinal tract by no means indicates that this number complained of gastrointestinal symptoms. Although the liver contained tubercles in 44.4 percent of patients, it is doubtful that any known liver function test would have revealed impaired hepatic function in any of these patients.

Our findings support those of Pinner (*1*) who states:

"* * * The vast majority of infection is by the aerogenous route. * * * Ghon and Kudlich reported in 1930 the primary complexes in over 2,000 cases. The lung was the site of primary infection in 96 percent of the cases, the intestinal tract in 1.14 percent, the skin in 0.14 percent, nose, tonsils, parotid gland, middle ear, and conjunctiva, each in less than 0.1 percent.

"* * * Evidence of hematogenous spread can frequently be found, particularly in the terminal phases, but if present, such spread is, as a rule, clinically insignificant and most often demonstrable only by microscopic or bacteriological study."

Aronson (*2*) points out that the military death rates cannot be properly compared with those for civilians. The death rate for tuberculosis in the Army is about 7 per 100,000 as compared with 48 per 100,000 for the United States as a whole. The number of deaths by age groups is weighted by the preponderance of men between 20 and 30 years old in the Army. Aronson shows an even greater preponderance in this age group than we do, probably because of our pre- and post-war load of veterans of World War I, and the fact that his figures probably included young patients too ill to be transferred to Fitzsimons Army Hospital from the various station hospitals. By means of comparing death rates for tuberculosis with average age distribution for the Army as a whole, he determined that the death rates in the Army increase with age in a manner similar to that seen in the civil population. Forty-three percent of his patients were Negroes as compared with 24 percent of ours. From this we may surmise that many of his Negro patients died before they could be transferred to this hospital. The percent of Indians in his series was 2.7, almost identical with ours. We agree with his statement: "There was no sharp racial variation in the percentage of deaths at different ages."

There is little uniformity in the literature regarding the duration of symptoms. Rogers (*3*) found that the mean duration of tuberculosis was 13 months for Negro and 18 months for white patients. Opie (*4*) observed that among Negroes living in Jamaica, the duration of tuberculosis for those between 15 and 30 years of age averaged 9 months, while for a group of white patients of comparable age in Philadelphia it averaged 28 months. Pinner and Kasper (*5*) found that tuberculosis averaged 33.2 months for 96 whites and 10.8 months for 47 Negro patients. Aronson found the average for white to be 5.7 months, while that for the Negro patients was 6.1 months. His explanation for his disagreement with other writers was the probability that his cases were of a more acute type. Our average durations of 4 years and 6 months for white and 1 year and 7 months for Negro patients do not seem unreasonable in view of the nature of the group studied. Rubin (*6*) supports our impression that it is difficult to state what degree of hematogenous spread constitutes classic miliary tuberculosis. He classifies hematogenous spread as miliary generalized acute, gradual slow, or intermittent seeding.

Empyema as a complication of pulmonary tuberculosis is well covered in the literature. We found nothing startling about the incidence of this complication.

Pulmonary hemorrhage as a cause of death in phthisis is generally minimized. Pinner (*1*) states: "Only a very small percentage of hemorrhages are immediately fatal by exsanguination or asphyxia. * * * Sudden death during hemorrhage is more often caused by asphyxiation than exsanguination." Our findings of hemorrhage as the immediate cause of death in 10.8 percent of the patients suggests that this complication is more serious than has been supposed.

Cor pulmonale as a complication of pulmonary tuberculosis was studied by Kuraner and Webb (*8*). Their material was drawn from the present series of 500 patients. They agreed with others as to the frequency of emphysema and fibrosis in chronic cor pulmonale in tuberculosis.

Tuberculous dissemination.—Rich (*9*) drew attention to the difference in the degree of involvement of different tissues by *Mycobacterium tuberculosis.* He stated:

"In the human being progressive, destructive lesions arising in the tissue in question (i. e., lesions not resulting from extension by direct continuity from contiguous susceptible organs) are familiar, for example, in the lung, kidney, intestine, fallopian tube, epididymis, prostate, adrenal, bone, brain, skin, eye, and lymph nodes. They are rare, however, in the skeletal muscles, pancreas, thyroid, heart, liver, spleen, ovary and testis."

Miliary tuberculosis was found in 11 patients in our series but hematogenous spreads occurred in many others, possibly as a terminal or preterminal event. Our findings of only 4.2 percent with involvement of the bone marrow is at variance with Rich's experience as indicated in table 7. In our series the liver was found to be more frequently involved than the spleen. The adrenals contained tubercles in 14 percent of the patients but the involvement was not extensive. Intestinal involvement was more often associated with positive sputum than with hematogenous spread. Eye involvement was negligible and skin involvement was found in only 0.9 percent. Our finding of 5 percent involvement of the thyroid and 1.8 percent hematogenous involvement each of myocardium and pancreas is greater than table 7 would indicate. Our figures agree remarkably well with those of Aronson even though he included all deaths from tuberculosis and did not make primary pulmonary tuberculosis a criteria for study as we have done. These findings have strengthened the belief that tuberculosis in the United States is usually primarily pulmonary in origin.

TABLE 7.—*Degree of involvement of different human tissues in miliary tuberculosis, compared with their tendency to be the site of extensive local lesions* [1]

Tissue	Always containing innumerable tubercles in miliary tuberculosis	Frequently the site of extensive lesions [2]
Lung, marrow	±	+
Spleen, liver	±	0
Kidney, adrenal, lymph nodes, brain, skin, fallopian tube, prostate, epididymis, intestine, eye	0	+
Heart, skeletal muscle, testis, ovary, pancreas, thyroid, parathyroid, hypophysis, stomach	0	0

[1] Rich, A. R.: The Pathogenesis of Tuberculosis, Charles C Thomas, Springfield, Ill. 1944. p. 318.
[2] I. e., lesions arising in the tissue, not extending into it by direct continuity from an adjacent susceptible tissue.

Gastrointestinal and peritoneal.—Goldberg (*10*) in a study of 230 autopsies of adults with tuberculosis found that 180 or 80 percent showed evidence of ulcerative enterocolitis and that this complication was more frequent in patients with advanced caseous pneumonic tuberculosis. The incidence in his series was higher than that in ours (50 percent), probably because we had an appreciable group (14 percent) without any cavities, and also a large group with a fulminating process who died within less than 1 year from the onset. Our impression was in agreement with Goldberg's that the order of frequency of these lesions was cecum, ileum, colon, and that anorectal tuberculosis occurs in only about 4 percent of patients dying with pulmonary tuberculosis. Rubin (*11*), in a study of 500 consecutive autopsies on patients who died of pulmonary tuberculosis, found macroscopic tuberculous ulcerations in 324, an incidence for the entire group of 65 percent. The

average duration of illness in his series was over 6 years, as compared to 4½ years in our cases. This accounts for the higher incidence in his series. In the majority of his cases the course of the disease was chronic or subacute, while in most of ours it was acute.

Goldberg, Rubin, and others quoted by them, found a higher incidence of gastrointestinal ulceration than we did because their cases were of longer duration. This explanation, however, is inconsistent with our finding of a higher incidence in our cases of short duration. We are unable to account for this inconsistency. On the other hand, Rubin found intestinal tuberculosis in 78 percent of his patients under 31 years of age as compared with 39 percent in those over 50 years of age, a finding with which we agree.

Crawford and Sawyer (*12*) in a series of 950 cases, which at autopsy showed some form of pulmonary tuberculosis, found 645 with gastrointestinal involvement and 563 with definite ulcerative lesions. They also found the greatest number of deaths in the 25 to 35 years of age group (50 percent of their total). They found the highest incidence of tuberculous enteritis in the cases of short duration. It is possible that modern methods of treatment, such as early pneumothorax, have reduced the incidence of intestinal tuberculosis. According to Koenig (quoted in (*10*)) tuberculous peritonitis is associated with lung involvement in 92 percent of cases. In death from tuberculosis of other organs, the peritoneum at autopsy is found to be involved in from 10 to 15 percent of the cases. In our series, involvement of the peritoneum was tenth in frequency of involvement of organs, or lower in frequency than indicated by Goldberg. Fishberg (*13*) states that peritoneal involvement is most common in miliary cases, and that only a few result from intestinal perforation. This is at variance with Buchbinder's statement (quoted by Goldberg (*10*)): "While blood-borne infection is to be considered, it would appear that involvement of the peritoneum is by way of infected peritoneal covered structures. A notable exception is, of course, a blood-borne miliary tuberculosis." Our finding of 87 percent of the cases of tuberculous peritonitis associated with intestinal tuberculosis supports the latter opinion.

Upper respiratory tract.—Tuberculosis of the upper respiratory tract is generally considered to be secondary. Myerson (*14*) states that nasal, paranasal sinus, oral, and pharyngeal involvement are extremely rare. There were none in our series. The incidence of middle ear involvement varies with different authors. Thompson (quoted by Myerson) reported it in 0.43 to 1.82 percent of all tuberculosis cases. Aronson found it in 0.8 percent of his tuberculous patients in the Army; we found it in 1.7 percent.

Laryngeal tuberculosis appeared in 26 percent of our patients.

According to Myerson "the incidence of laryngeal tuberculosis varies with almost every writer," ranging from 1.2 to 97.4 percent with an average of 14 percent. He found the highest incidence in the group between 20 and 40 years of age. Lederer (quoted by Goldberg) and Myerson (*14*) stated that the frequency of involvement increases with the duration of the pulmonary disease to which it is almost invariably secondary. In our series there is a definite correlation between laryngeal tuberculosis and pulmonary cavitation, and also between laryngeal and intestinal tuberculosis which was also present in a higher percent in the patients with cavitation. Myerson reported tracheobronchial tuberculosis in 4 percent of all tuberculous patients, with women outnumbering men 3 to 1. In our series (all men) 14 percent had this complication.

Genitourinary tract.—McKenna (quoted by Goldberg (*10*)) points out that the incidence of genitourinary tuberculosis depends on the type of patient. He quotes Hubner as finding that 3 to 5 percent of patients with pulmonary tuberculosis have this complication; however, it is found in from 50 to 70 percent of those with extrapulmonary tuberculosis. Our findings of kidney involvement in 29.2 percent and genital involvement in 15 percent indicate that such lesions are commoner than is generally believed. Aronson found an incidence of 39.4 percent with kidney involvement and 30 percent with genital involvement among deaths from tuberculosis of all types. The hematogenous route is primarily implicated in the spread and the highest incidence of urogenital tuberculosis is found in patients 20 to 50 years of age. The duration of the infection seemed to have little effect on the incidence of this complication.

Endocrine glands.—Aronson found tuberculosis of the thyroid in 3.9 percent of his tuberculous patients at autopsy. Our 5 percent may have resulted from terminal hematogenous spread. Although Addison's disease is rare among patients with pulmonary tuberculosis, adrenal involvement may be an important factor in the terminal cachexia of such patients (*15*). Aronson found such involvement in 25 percent of his series. We found tuberculosis of one or both adrenals in 14 percent of our series. We did not find any case of tuberculosis of the pituitary body.

Liver, spleen, and pancreas.—Because the liver and spleen are seldom mentioned in the literature on tuberculosis, our finding of 44 and 41 percent involvement, respectively, was surprising. Aronson, in his series, found 66 percent involvement of both. The involvement usually consisted of miliary tubercles which suggests a terminal or near terminal hematogenous spread of little clinical significance. The pancreas, renowned for its resistance to tuberculosis was found to contain tubercles in 1.8 percent of our series.

Central nervous system.—Although tuberculomas and tuberculous meningitis are extensively covered in the literature, most statistics include infants and children and cannot properly be compared with our findings. Furthermore most studies have been based on total deaths from tuberculosis rather than deaths with a primary or apparently primary pulmonary infection. Aronson found cerebral tuberculosis in 12 percent and meningitis in 36 percent of his series.

Bone tuberculosis is usually reported in series that include children. Our finding of 4.2 percent with bone involvement compares favorably with that of Aronson (5.6 percent) although his series included a higher proportion of Negroes.

Heart.—Although tuberculous pericarditis, according to authors quoted by Goldberg, was found in 6.3 percent of 1,780 autopsies on tuberculous patients, it was diagnosed in only 0.4 percent of 7,646 collected cases. Aronson found it in 2.1 and we found it in 3.2 percent. Strauss (quoted by Goldberg) found that tuberculosis of the myocardium is rare, varying from 2.64 percent in children to 0.33 percent in adults with tuberculosis who are examined at autopsy.

Skin tuberculosis is rare in America but internists are always on the lookout for cutaneous manifestations with pulmonary tuberculosis (*16*).

Clubbing of the fingers was found in 22 percent of our cases. According to Mendlowitz (*17*) clubbing is most pronounced in chronic suppurative conditions such as bronchiectasis and empyema and is seen less frequently in pulmonary tuberculosis, and then usually in protracted cases. Our observation of clubbing in many cases of short duration (22 percent in cases of less than 1 year's duration) was surprising. It is doubtful that hospitalization at a high altitude (Denver, Colo.) played any appreciable part in the development of this finding. The duration of the pulmonary disease seemed to have no appreciable effect, but the incidence of clubbing reached 41 percent of those who died after attaining the age of 50 years, when bronchiectasis was probably more prevalent. There was no significant correlation between clubbing and empyema. We did not check the relation of clubbing with amyloidosis because of the small number of patients with amyloid disease.

Amyloidosis.—Wassersug (*18*) states that chronic suppurative disease, tuberculous or otherwise, usually leads to amyloid disease. Although few of our patients were classed as chronic, we found 16 percent with amyloidosis among those who died after an infection of 3 years' or more duration. The actual age made relatively little difference in the incidence other than to reflect the duration of the illness. Crawford and Sawyer working in this hospital studied a large group

of cases prior to 1934 and found amyloidosis in 15 percent. Cohen (*19*), in 143 autopsies of tuberculous patients, found amyloidosis in 39 percent, 19 percent of whom had coexisting empyema.

SUMMARY

In a series of 500 autopsies of patients in whom pulmonary tuberculosis was the primary cause of death, the following exceptions to the currently accepted figures were noted:

1. Pulmonary hemorrhage was the apparent cause of death in 10.8 percent of our series and consequently cannot be considered a benign complication as some suppose. The clinician should be prepared to treat it actively, if necessary.

2. Enteric and laryngeal tuberculosis appeared in a higher incidence in cases of short duration than was generally reported in the literature. We found a higher incidence in cases of less than 5 years' duration than in more chronic cases, and a higher percent in young adults than in older age groups.

3. The tracheal involvement paralleled the enteric and laryngeal involvement and was found in a higher percent (14) than was generally reported.

4. Genitourinary infections in men as reported by Aronson (*2*) and us were found in a higher proportion of cases than has generally been suspected.

5. The adrenal glands revealed seeding, possibly from a terminal hematogenous spread, in a higher proportion than was generally reported.

6. Clubbing of the fingers was found in many cases of short duration.

7. Amyloid disease as a complication of pulmonary tuberculosis seems to be disappearing.

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Abdominal Hysteropexy for Uterine Prolapse

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THE literature is replete with various types of uterine suspension, as well as methods for the correction of prolapse. Perhaps the most widely used procedures for prolapse during the childbearing period is the Manchester or Fothergill operation and various modifications of it. Although pregnancy and delivery do occur thereafter, abortions and severe lacerations of the remaining cervix are complications to watch for. The main supports of the uterus are the structures which are encompassed by a plane about the cervix and upper vagina. It occurred to the senior author about 12 years ago that these supports could be strengthened by the abdominal approach without amputation of the cervix and that this would give a much better chance for normal pregnancy and delivery to follow. Therefore, the following technic was developed and has proved satisfactory in such cases.

Operation.—The abdomen is opened through a midline incision and the uterus is lifted up with a ligature through the fundus. A silk ligature is then passed from outside the uterosacral ligament one-third to one-half the distance toward the sacrum. It then picks up the peritoneum and rectovaginal fascia in the cul-de-sac. If the fascia is badly stripped away it may be necessary to open the peritoneum in the cul-de-sac to locate and pick up the fascia in this area. The ligature is then passed through the back of the cervix near the vaginal reflection. It dips again to pick up the cul-de-sac as before, and then passes out through the uterosacral ligament on the opposite side. The ligature is then tied. Another ligature is taken through the uterosacral ligaments and cervix above this one and tied. Thereafter, ligatures are passed through one ligament, the cul-de-sac fascia and peritoneum,

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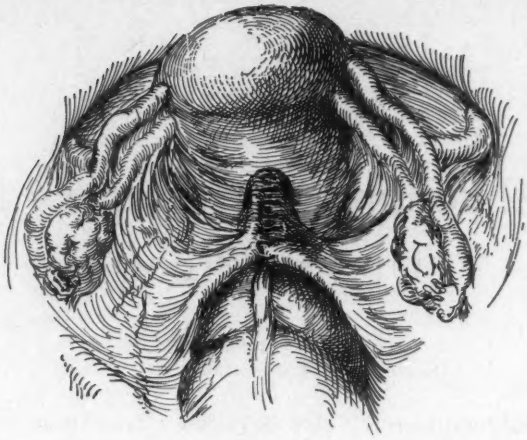
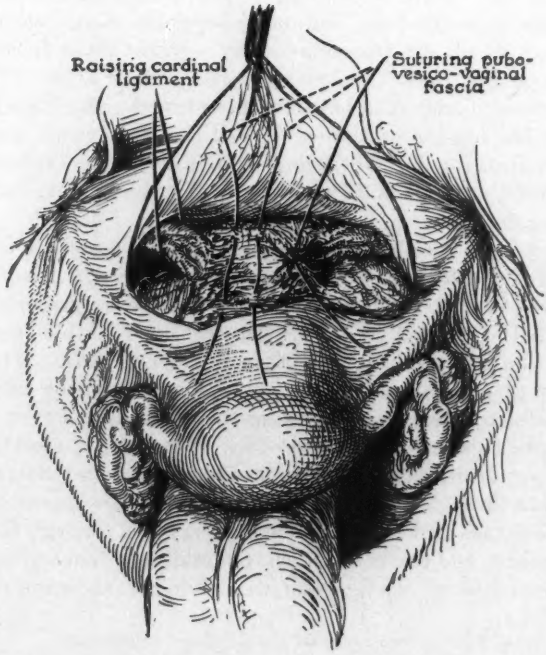


Figure 1.



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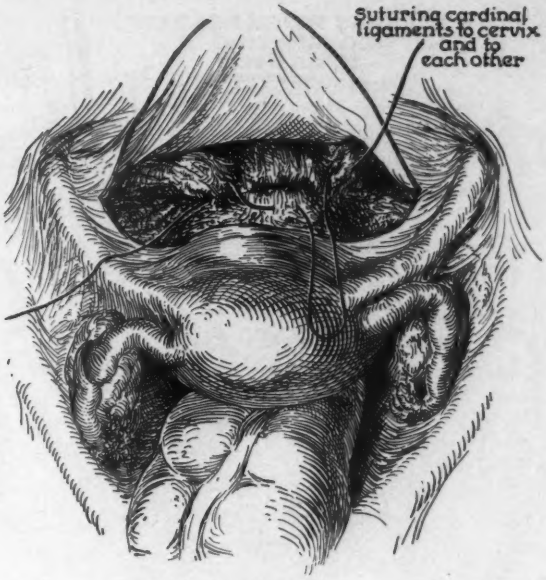

Figure 3.

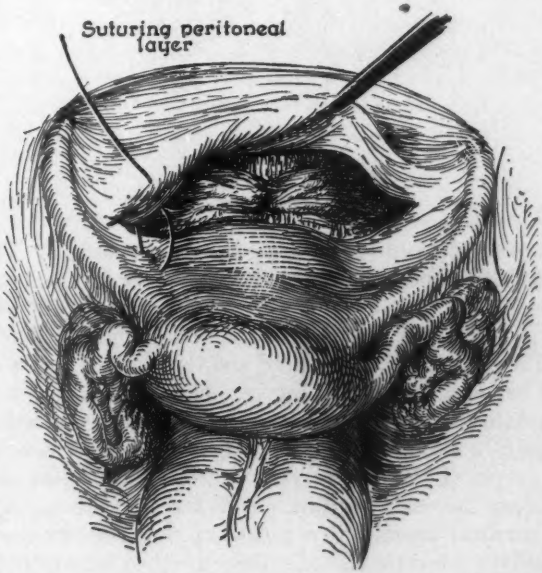

Figure 4.

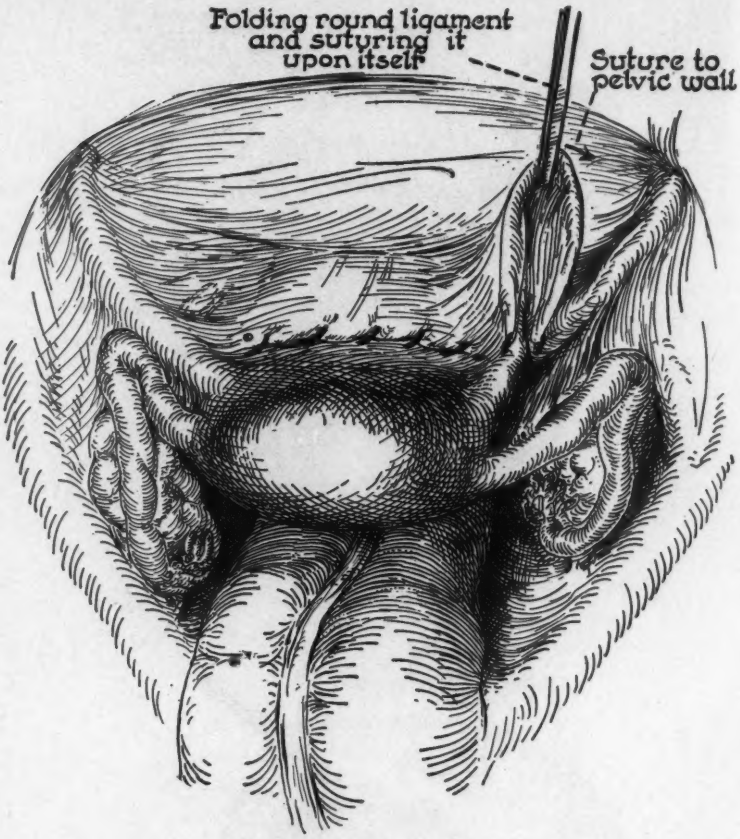

Figure 5.

out through the opposite uterosacral ligament, and tied. These ligatures are continued every one-fourth inch back to the front of the rectum and made snug around it to help maintain the "lift" which has been given to it by bringing up the fascia (fig. 1). The uterus is then held toward the patient's head and the bladder flap is depressed to a level below the tip of the cervix. The pubovesicocervical fascia can be visualized and usually the rent in it stands out well. Silk is used to place a suture through the edges of the rent and to anchor it to the cervix at the uterocervical juncture. Sufficient sutures are placed to close the rent and to anchor the fascia to the cervix wall (fig. 2). The cardinal ligaments are picked up near the cervix with Allis' forceps and the relaxation noted. These are then sutured to the cervix and to each other at the uterocervical juncture and downward on the

cervix, placing sutures through fascia and cervix and bringing the ligaments together. This forms a sling across the front of the cervix to give good support as well as proper tilt to the fundus (figs. 3 and 4). The fascial repair obliterates the cystocele. The bladder is then replaced at a slightly higher level than formerly as the fascial repair has definitely lifted it up. Plain catgut is used to fix the bladder in place. Silk sutures are used to triplicate the round ligaments on themselves (fig. 5). The suture through the outer "knuckle" catches the fascia at the internal ring as well as the distal part of the round ligament, whereas the medial "knuckle" is anchored well into the base of the round ligament. The abdomen is closed in layers.

Danger points.—(1) When picking up the uterosacral ligaments in the back, watch for low-placed ureters caused by lower position of the bladder. (2) Anteriorly when picking up the cardinal ligaments insert the needle through them parallel to the course of the ureter, being careful to stay medial to it. (3) When triplicating the round ligaments, be sure no holes are left through which a piece of bowel may work its way.

DISCUSSION

It may be seen from the preceding that this procedure will correct the prolapse and associated cystocele and rectocele. It does not correct gaping of the vaginal orifice, nor the defect in the pelvic floor which exists concomitantly in many of these cases. If any one of these three conditions is associated with the prolapse it has to be corrected from below. If the elongated cervix is amputated it is only a partial amputation and is not fraught with the same dangers as in the Manchester procedure. This complication is seldom seen as a prolapse is not usually neglected sufficiently to allow it to develop. In half of the patients with prolapse today the pelvic floor and introitus have been adequately repaired following delivery and, therefore, only the abdominal procedure need be carried out. This procedure has been carried out successfully in two virgins with third-degree prolapse.

Application of Civil Defense Lessons Learned in World War II[1]

WILLIAM L. WILSON, *Colonel, MC, U. S. A.*[2]

WHAT is to be said here is not officially proposed as a guide; it must not be construed in any manner to have military or other governmental sanction or approval. It does not convey official decision or policy; this applies especially to the suggested applications of the lessons which are solely my own responsibility. This subject has so many possibilities that it has not been easy to outline, or direct into an application of past experience, the lessons we should have learned. I shall explore this difficult subject by listing some of the lessons which were learned in World War II, examine them briefly, and then consider the manner in which they might be applied to future planning. As we review them be reassured that a necessary stressing of the disasters of war in nowise predicts war soon or in the distant future.

LESSONS

1. *The United States is probably the most changing and shifting nation in the world.*—Like others, we have a population that is aging[3,4] although increasing rapidly. It has a larger proportion of women than men for the first time.[5] Employment has adapted to industrialization at a rapid rate. This has accompanied urbanization[6] (fig. 1) and steadily decreasing agricultural employment until there are now 50 cities with more than 200,000 population, a minimum likely to be considered good atomic bomb targets.

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[1] Condensation of a paper delivered 20 February 1950 before the Tenth Annual Congress on Industrial Health of the American Medical Association, New York, N. Y. The full text can be obtained from the Technical Information Office, Surgeon General's Office, Department of the Army.

[2] Office of the Surgeon General, Department of the Army.

[3] WHELPTON, P. K.; ET AL.: Forecasts of the Population of the United States 1945–1975. United States Government Printing Office, Washington, D. C., 1947.

[4] National Resources Committee: The Problems of a Changing Population. United States Government Printing Office, Washington, D. C., 1938.

[5] FURMAN, B.: Women to exceed men in 1950 census. The New York Times, New York, N. Y., Feb. 13, 1950. p. 23.

[6] REED, L. J.: Changing problems growing out of change in composition of population. Am. J. Pub. Health 38: 160–163, Jan. (pt. 2) 1948.

A quick look at our past may indicate some of the problems of the future. For example, in the recent war, we had a voluntary migration which caused great difficulty in furnishing health services at home. We may visualize major problems in administration of governmental, industrial, and voluntary health services,[7] when more than half of our population is mobile. In the 7-year period 1939–46, 70 million of our people moved from one house to another at least once.[8] Of those who moved, half moved to a different State. Thirty percent of our 14 million World War II veterans moved in 1947. Our West-

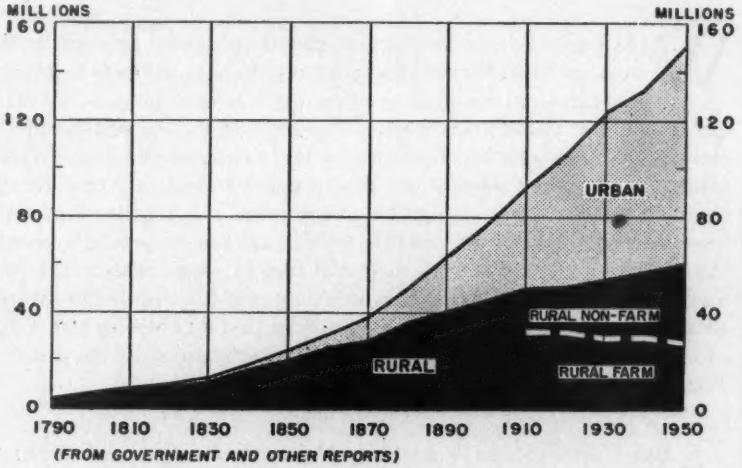

Figure 1.—Population changes in the United States by residence areas and time periods.

ern States nearly doubled their population while those in the East and the Midwest had losses. Most of this accompanied occupational, vocational, or economic relocation in adjustment to a shifting of industry and industrial facilities. During the war there was a pronounced shifting in type and volume of production to items for military use.

During and since the recent war statistics have purported to prove widespread physical unfitness, critical health hazards, inadequate health services, or excessive wastes of medical means incident to the war. Although a maximum number of our physicians was with the

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[7] BLANKENSHIP, C. F., and SAFIER, F.: A Study of Medical Problems Associated With Transients. United States Public Health Service Bulletin No. 258, United States Government Printing Office, Washington, D. C., 1940.

[8] Bureau of the Census, Department of Commerce: Current Population Reports, Series P–20, No. 14: Internal Migration of the United States, April 1940 to April 1947. Department of Commerce, Washington, D. C., Apr. 15, 1948.

armed forces in 1945 when 60,721 (31.4 percent of total) were on duty,[9] American community health and necessary medical services survived the war with remarkable success in this country.[10] At the same time the armed forces and occupied areas had the best medical services in history. The furnishing of health services to such a mobile civil population in this country has not, however, been directly hampered by enemy action in the past. If we add the possibilities of enemy action against our civilians, we realize that our efforts will be inadequate unless future obligations are met in a more energetic, intelligent, and integrated manner than ever has been known. During World War II our lack of coordination of medical activities and confusion in integrating administration of military and civilian health affairs was so complex it can be shown best by diagrams (figs. 2 and 3).

What were some achievements of American medicine in World War II? After 7 years beginning with 1940, only 5 percent of all live births were unattended by physicians. Live births occurring in hospitals increased by 46 percent. Infant mortality rates dropped by 25 and maternal mortality rates by more than 50 percent. During this critical period the number of nurses consistently increased in United States hospitals by nearly 33 percent. While unexcelled medical and nursing attendance was being rendered 14 million in our armed services throughout the world, mortality at home was decreasing as was morbidity from communicable and other diseases. Army medical officers away from the United States have recently supervised the re-establishing of health services for 140 millions in Europe,[11] for 80 million Japanese, and for the inhabitants of the Philippines and Korea. They also supervised the health of 16 million migrants from 32 countries as the latter resettled in Europe before 1947, as well as 6 million Japanese during repatriation from 19 Asiatic-Pacific areas.

In view of the facts cited, it might be advantageous to an enemy to direct his weapons at our industrial installations rather than at our armed forces.[12] With this in mind the National Security Resources Board has proposed maximum practicable dispersion of industry.[13] The strategic significance of industrial-plant location in the event of

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[9] Personnel and Administration, General Staff, Department of the Army: Data furnished by communication dated 27 October 1948.

[10] WILSON, W. L.: The Value of a Physician in War. Address before Nassau County Medical Society, Garden City, N. Y., Mar. 29, 1949.

[11] Supreme Headquarters, Allied Expeditionary Force: Technical Manual for Public Health Officers, Military Government for Germany. Feb. 1945.

[12] MANSON, E. S.: American Security and Access to Raw Materials. World Politics I (2): 147, Jan. 1949.

[13] National Security Resources Board: National Security Factors in Industrial Location. United States Government Printing Office, Washington, D. C., Sept. 1948.

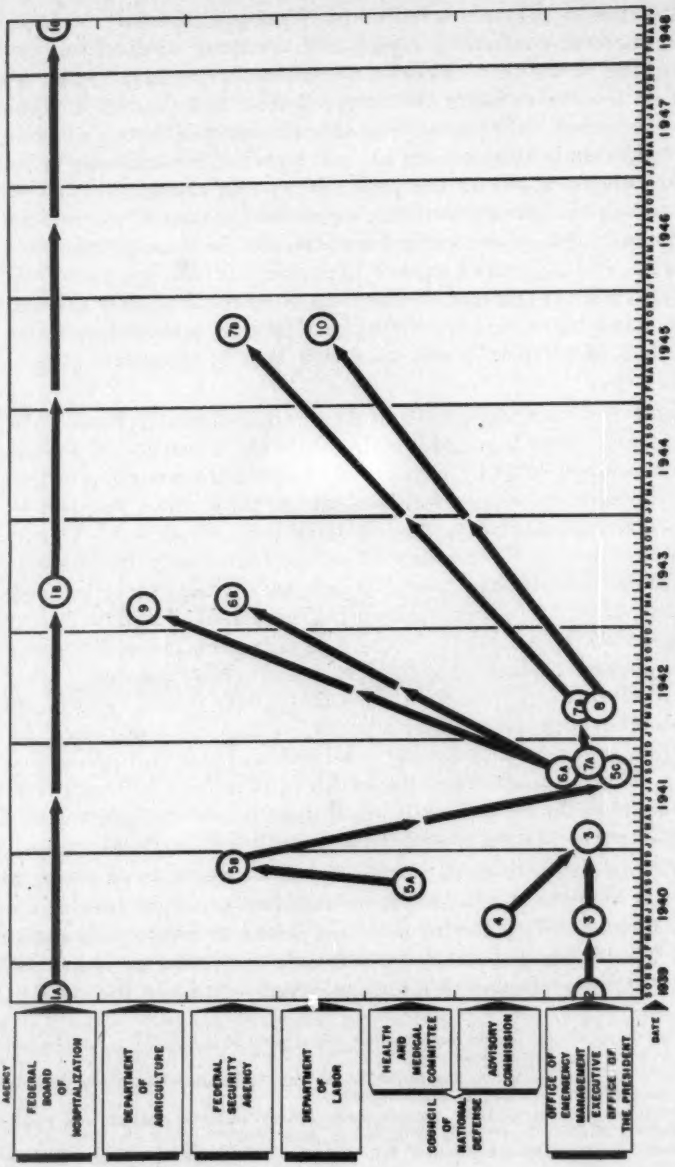

Figure 2.—Coordination of medical affairs—general organization.

The following is supplemental to the chart on the opposite page:

1. Federal Board of Hospitalization:

a. Organized 1 November 1921 to coordinate hospitalization activities of the Army, Navy, Public Health Service, Veterans' Administration, St. Elizabeths Hospital, and Office of Indian Affairs.

b. Designated advisory agency to the Bureau of the Budget by Budget Circular 419, 7 May 1943.

c. Terminated 30 June 1948 by letters from the director, Bureau of the Budget to members of the Board, dated 28 May 1948.

2. Executive Order 8248; 8 September 1939, authorized establishment of an office for emergency management.

3. Administrative order of the President, 25 May 1940, set up office for emergency management in the Executive Office of the President (page 2109, Federal Register of 4 June 1940) contained only Office of Defense Transportation and the Philippine Alien Property Administration on 30 June 1948, former to terminate 28 February 1949.

4. Advisory Commission of the Council of National Defense set up 29 May 1940; funds available thereto reallocated to the Office for Emergency Management by the President, 28 February 1941.

5. Health and Medical Committee:

a. Of the Council of National Defense, established 19 September 1940.

b. On order of the Council of National Defense, approved by the President 28 November 1940, transferred to Federal Security Agency.

c. Transferred to Office of Defense Health and Welfare Services, Office for Emergency Management, Executive Office of the President, by section 6, Executive Order 8890, 3 September 1941.

6. Office of Defense Health and Welfare Services:

a. Established in the Office for Emergency Management, Executive Office of the President by Executive Order 8890, 3 September 1941, to coordinate all health, medical, welfare, nutrition, recreation, and other related activities affecting the national defense, including those aspects of education under the Federal Security Agency.

b. Abolished by Executive Order 9338, 29 April 1943 and all functions transferred to Federal Security Agency.

7a. The President's approval of a letter of 30 October 1941 from the Administrator, Federal Security Agency, established the Procurement and Assignment Agency in the Defense Health and Welfare Services.

b. Procurement and Assignment (office) transferred to War Manpower Commission by section 4, Executive Order 9139, 18 April 1942.

8. War Manpower Commission established in the Office for Emergency Management by Executive Order 9139, 18 April 1942, to assure most effective mobilization and utilization of manpower for war.

9. By Executive Order 9130, 3 March 1943, functions, powers, and duties with respect to nutrition transferred from the Office of Defense Health and Welfare Services to the Secretary of Agriculture.

10. War Manpower Commission terminated by Executive Order 9617, 19 September 1945, and all functions transferred to Department of Labor except procurement and assignment service (established as agency and transferred without official change of designation), which was transferred to the Federal Security Administration.

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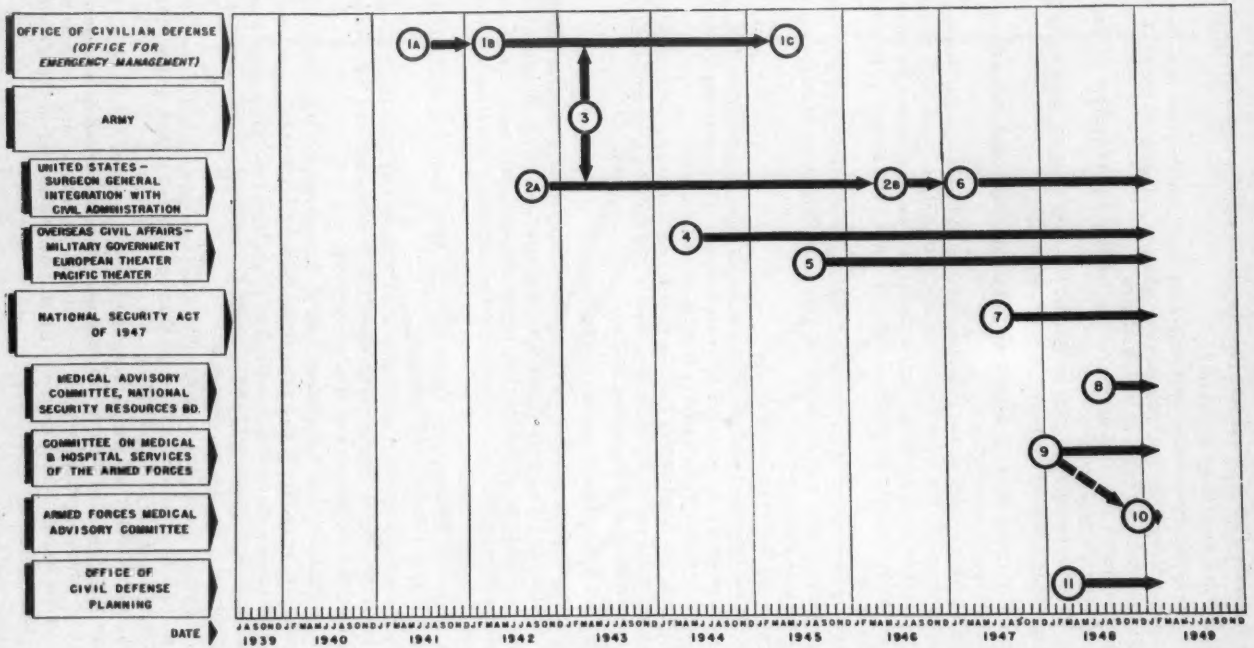
OFFICE OF CIVILIAN DEFENSE (OFFICE FOR EMERGENCY MANAGEMENT)
ARMY
UNITED STATES – SURGEON GENERAL INTEGRATION WITH CIVIL ADMINISTRATION
OVERSEAS CIVIL AFFAIRS – MILITARY GOVERNMENT EUROPEAN THEATER PACIFIC THEATER
NATIONAL SECURITY ACT OF 1947
MEDICAL ADVISORY COMMITTEE, NATIONAL SECURITY RESOURCES BD.
COMMITTEE ON MEDICAL & HOSPITAL SERVICES OF THE ARMED FORCES
ARMED FORCES MEDICAL ADVISORY COMMITTEE
OFFICE OF CIVIL DEFENSE PLANNING
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1939 1940 1941 1942 1943 1944 1945 1946 1947 1948 1949

Figure 3.—Medical experience in United States integration of civil and military health administration, 1939–49.

The following is supplemental to the chart on the opposite page:

1. Office of Civilian Defense:

a. Established in Office for Emergency Management by Executive Order 8757, 20 May 1941; amended by Executive Orders 8799, 20 June 1941, and 8822, 16 July 1941.

b. Amended by Executive Order 9134, 15 April 1942, which set up a civilian defense board.

c. Abolished by Executive Order 9562, 4 June 1945, effective 30 June 1945.

2. Surgeon General of the Army:

a. Charged with administration of military hospitalization and evacuation operations (SPOPH 322.15—15 September 1942, Headquarters, SOS, Washington) which provided for coordination with and mutual assistance in civilian defense.

b. Operations continued but no further civilian defense aspects as of 30 June 1945.

3. By mutual agreement and directives from Army and Civilian Defense Offices, mutual support through Army staffs and T/O units and civilian defense affiliated units and mobile teams developed in spring 1943.

4. Civil health affairs established as a major function in Supreme Headquarters, Allied Expeditionary Force, 8 May 1944, and continued through United States headquarters to date.

5. Civil health affairs established as a major function in Headquarters, Supreme Commander, Allied Powers in Pacific, summer 1945.

6. Surgeon General of the Army directed development of study on integration of administration of civil with military health affairs, March 1947.

7. National Security Act of 1947 set up agencies for administration and integration of civil and military plans and operations, 26 July 1947 (Public Law 253, Eightieth Congress).

8. Medical Advisory Committee established within the National Security Resources Board by the chairman, announced 29 July 1948.

9. Committee on Medical and Hospital Services of the Armed Forces (Hawley) established by the Secretary of Defense, in a letter dated 1 January 1948.

10. Armed Forces Medical Advisory Committee (Cooper) established by the Secretary of Defense, announced 12 December 1948.

11. Office of Civil Defense Planning established by the Secretary of Defense in a letter dated 27 March 1948.

another war was cited. Many factors governing the economic evaluation of plant locations were listed by the Board, but there was no mention of health and medical service factors. These were deemed of maximum importance by the United Kingdom, for the logic of which we may go on to other lessons.

2. *National patterns were developed during World War II by the United States, the United Kingdom, Japan, and Germany* (in the order of their success in escaping direct damage by enemy actions).—These are summarized in table 1.

TABLE 1.—*Industrial-medical preparedness patterns developed during World War II*

Activity considered	United Kingdom	United States	Japan	Germany
Succeeded in national combat efforts	Yes	Yes	No	No.
Continued as economic and political unit	Yes	Yes	Yes	No.
Homeland attacked by enemy	Yes	No	Yes	Yes.
Mobilized armed forces at earliest practicable time.	Yes [1]	Yes	Yes	Yes.
Increased armed force mobilization progressively	Yes	Yes	Yes	Partially.
Developed and expanded effective civil defense at earliest practicable time.	Yes	No	No	No.
Contained capacity for increasing productive output at beginning.	Yes	Yes	No	Yes. [2]
Shifted economic and industrial effort to prosecution of war at earliest practicable time and to maximum degree.	Yes [3 4]	No	No	No.
Mobilized own maximum armed strength supportable by total economic and industrial potential.	Yes [3 4]	No	No	No. [5]
Increased progressively home-front participation by women, noncombatant men, and the physically handicapped.	Yes [4]	No	No	No.
Applied maximum practicable war production capacity of women.	Yes	No	No	No. [6]
Estimated situation with reasonable accuracy	Yes [7]	Partially	No [8]	No.
Planned ahead on realistic long-range basis	Yes	No	No [8]	No. [8]
Coordinated management of available resources	Yes	No	No	Partially.
Integrated military and civilian use of resources	Yes	No	No	No.
Observed by scientific method the effects of prescribed policies on living conditions and use of manpower.	Yes	No	No	Partially.

[1] The Prime Minister and the Minister of Defense: Strength and Casualties of the Armed Forces and Auxiliary Services of the United Kingdom 1939 to 1945. London, H. M. S. O. (Cmd. 6832), 1946.
[2] United States Strategic Bombing Survey: The Effect of Bombing on Health and Medical Care in Germany. United States Government Printing Office, Washington, D. C., 1945.
[3] United States Strategic Bombing Survey: Data from various reports of the group.
[4] The Minister of Labour and National Service: Committee on Skilled Men in the Services: Second Report and a Memorandum by the War Office, London, H. M. S. O. (Cmd. 6339), 1942.
[5] United States Strategic Bombing Survey: The Effects of Strategic Bombing on the German War Economy. United States Government Printing Office, Washington, D. C., 1945.
[6] In the early years German war production was not limited by her war potential or resources but by demand, that is, by the notions of the German war leaders as to what was required for achieving their aims (fig. 4). The Germans did not plan for a long war, nor were they prepared for it.
[7] Churchill, W.: The Second World War, The Gathering Storm. Houghton Mifflin Co., Boston, Mass., 1948.
[8] United States Strategic Bombing Survey: Summary Report (Pacific War). United States Government Printing Office, Washington, D. C., 1946.

3. *A clear pattern of certain essentials must be preplanned and assured if a modern nation is to survive the disasters of war.*—In order of importance they are water and food; fighter aircraft or defensive guided missiles; coal, liquid fuels, and lubricants; transport by ground, sea, and air; communications; iron, and steel; other metals; certain chemicals; and medical material and services.

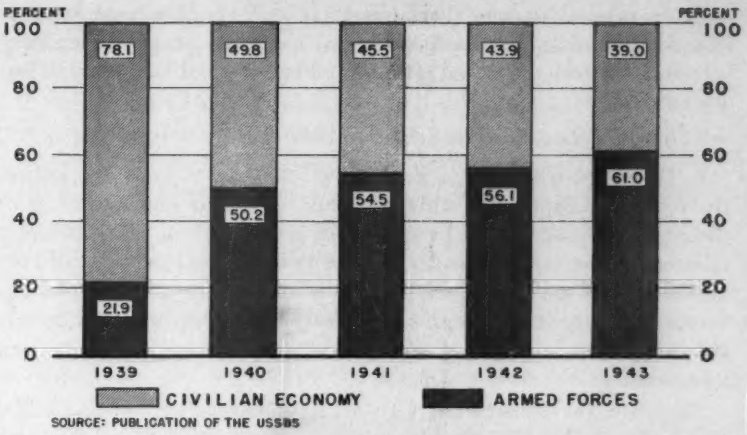

Figure 4.—German percentage distribution of industrial labor force producing for civilian economy and for armed forces as of May 31, 1939–43.

4. *Success in modern war requires maximum national mobilization and performance from the first intimation of war.*—This involves coordination of unbelievable complexity and maximum integration of military and civilian plans and operations. Experiences of World War II indicate the necessity for: a. National service from the start. b. Maximum military mobilization of air, sea, and ground forces from the start, with such changes in strengths and missions allotted to each as may become necessary. c. Maximum diversion of the civilian economy necessary to (1) hold off the enemy, guided missiles, and other attacks; (2) attack and destroy or disperse enemy forces; (3) occupy, govern, and reestablish over the former enemy an acceptable, new, or revised government (primarily ground-force responsibility); and (4) protect this nation from any future repetition of war. d. Maintenance of national mobilization only so long after hostilities cease as may be necessary to: (1) control the former enemy (primarily ground-force responsibility, but involving sea and air forces to a maximum from time to time);[14] (2) reconversion of our own economy to peacetime pursuits; and (3) have power to bargain and establish acceptable international relations during the postwar era to prevent menacing international situations, and to assure arranging of desirable peace treaties involving former enemies, allies, neutrals, and others. e. Involvement of minimum practicable number of governmental agencies with necessary major efforts, whether they be

[14] A Study of Operation "Vittles." Aviation Operations, Vol. 11, No. 5. New York, N. Y., Apr. 1949.

military or civilian, and their coordination.[15] f. Maximum integration for planning, administration, and implementation of necessary activities between the armed services and between military and civilian authorities.[16]

5. *Every nation is vulnerable to attack by a powerful enemy.*[17-19]

6. *There are definite priorities for military missions of any nation,* the industrial support of which depends on a clear understanding of those priorities and of the programs for meeting them. The primary missions of any nation in order of priority are to: (a) prevent military attack against the homeland; (b) attack and subdue the enemy armed forces; (c) attack and subdue the enemy homeland; and (d) occupy the enemy homeland and govern the enemy civilian population as long as necessary.

Not only government but also management, labor, the industrial physicians, public health physicians, and private physicians must understand this matter sufficiently to permit proper training, use, and physical and mental protection of personnel. There can be no bargaining on three points: (a) A failure to guarantee success to our Armed Forces against enemy attacks will insure our ruin, regardless of the extent to which our civilian economy may be disrupted by war; (b) Allocation of certain means to civilian uses is as necessary to successful combat as is allocation to military uses; and (c) Proper balancing between civilian and military requirements is our objective.

7. *Repeated forceful enemy attacks against our civilian population will result ultimately in economic collapse, submission, then defeat, regardless of our successes in combat.*

8. *Enemy attacks against the homelands of three nations during World War II followed a pattern characterized by homeland capacities for resistance to attack.*—The pattern of collapse is: (a) loss of control of the air; (b) lack of coal; (c) lack of liquid fuels and lubricants; (d) insufficient transport and communications; (e) insufficient food; (f) lack of steel; (g) insufficient armaments and ammunition; (h) insufficient civilian supplies; (i) insufficient raw materials such as iron and light metals; (j) insufficient services and utilities; (k) lack of capital equipment; (l) insufficient skilled manpower; (m) in-

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[15] Armed Forces: Screen Report—Release 113, Industrial Mobilization, 1949.

[16] War Department: Military Hospitalization and Evacuation Operations. United States Government Printing Office, Washington, D. C., 1942.

[17] A Report by the President's Air Policy Commission: Survival in the Air Age. United States Government Printing Office, Washington, D. C., 1948.

[18] DEVAN, S. A.: Planning National Defense 1950 to 1970. The Library of Congress, Washington, D. C., Sept. 1949.

[19] BUSH, V.: Modern Arms and Free Men. Simon and Schuster, New York, N. Y., 1949.

sufficient total manpower; (n) insufficient construction capacity; (o) insufficient medical and health supplies; and (p) insufficient medical and health services.

9. *New weapons will appear.*[18 19]—At first, these will be those which can be produced quickly. Others will appear from time to time by extension of research and development during the war.

10. *New protective measures or means for counterattacks will be developed.*—As new offensive and defensive means develop, planning, strategy, tactics, and operations must change.

11. *The civil population and its industrial facilities must adapt to ever-changing circumstances just as readily as armed forces.*—Swift changes or shifts of persons and organized units of manpower (of both sexes) must be possible, as well as material, services, and technics.

12. *Complex modern weapons, modern manufacturing, and modern society necessitate a large and ever-increasing number of skilled and trained men and women.*[15 20]

13. *Modern war creates an ever-increasing demand for complex equipment and enormous quantities of supplies.*—As an example, a recent study by the staff of the Army Surgeon General revealed that the barest minimum of medical equipment and supplies necessary to save and maintain the life of one atomic explosion victim for from 3 to 6 days would weigh 50 pounds, displace 4.2 cubic feet, and cost at least $25 at current prices.

14. *Developing and improving military technics will compel expedited and expanded industrial developments.*[21 22]

15. *A competing nation must anticipate maximum adaptation of its internal economy to a potential theater of operations;*[23 24] the former will limit the latter's extent by the potential of economic resources, preplanned management,[25-28] technologic developments of industry;

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[20] United States Strategic Bombing Survey: The Effects of Bombing on Health and Medical Services in Japan. United States Government Printing Office, Washington, D. C., 1947.

[21] WILSON, W. L.: The Army Surgeon's Part in Current Biological Warfare Defense Planning, Nov. 8, 1949.

[22] WILSON, W. L.: Research Needed in the Field of Sanitation (particular attention to civilian war problems). Conference Notes, Sanitation Study Section, National Institutes of Health, United States Public Health Service, Jan. 28, 1949.

[23] WILSON, W. L.: Military Responsibility in Civil Public Health. Lecture V, Military Hygiene, United States Military Academy, Sept. 16, 1948.

[24] WILSON, W. L.: Medical and sanitary care of civilian population necessitated by attacks from hostile aircraft. Army M. Bull. (no. 60) pp. 63–107, Jan. 1942.

[25] WILSON, W. L.: Medical aspects of government services in time of national emergency. Bull. U. S. Army M. Dept. 8: 493–498, July 1948; J. A. M. A. 137: (Organization Sect.) 793–795, June 26, 1948.

[26] WILSON, W. L.: Medical plans for civil defense and disaster relief. U. S. Armed Forces M. J. 1: 462–475, Apr. 1950.

[27] WILSON, W. L.: Civil organization for health in time of war. Bull. U. S. Army M. Dept. 8: 778–789, Oct. 1948.

this involves maximum integration of military and civil administration.[28] [29]

DISCUSSION

To apply these lessons we should (1) make sure that we are ready at any moment should disaster strike; (2) be prepared to apply to maximum advantage all the means we shall have available and do this efficiently, economically, and immediately; and (3) ask ourselves whether our industries would be prepared if disaster should come. True disaster is ever-changing and outdoes itself during war. Therefore, any agreements we reach now might be obsolete at an early date unless we continue our attention to them. The seriousness of the situation we might have to face, should be a great stimulus to proceed with the task at hand without delay. The lessons should be so applied that we will guarantee avoidance of the disasters we have considered, or at least that we shall not be overwhelmed. We should and can guarantee both if we act promptly. Suggested answers to our question will be restricted to activities proper for industrial health personnel to undertake actively. Let us test every proposal for action by the British concept that the organization of war is largely a question of priorities; the first for whatever may be necessary to keep the machines of war in action, the second for making optimum use of available skills.

Although the following suggestions should be accepted and implemented only after careful consideration, action should be the rule and further discussion and delay avoided.

1. For an interim period, starting now:

a. Uniform professional advice on industrial health affairs concerned with disaster control should be promulgated to management, labor leaders, health staffs, governmental agencies, and other interested organizations. This is particularly applicable to necessary means, their availability, development of optimal industrial environments, health aspects of migrations and evacuations, and of relocation of plants, facilities, services, equipment, and personnel.

b. Every advantage must be taken of opportunities to learn the responsibilities and authorities of workers, agencies, and groups who will engage in the broad field of disaster control. In this connection full study should be made of the fact that the only States having specific legislation for civil defense in disaster operations prior to 1

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[28] WILSON, W. L.: Administration of civilian medical care in total war. Bull. U. S. Army M. Dept. 9: 107–110, Feb. 1949.

[29] WILSON, W. L.: Military-Civil Integration of Health Services for Disaster Control. University of California School of Public Health, Berkeley, Jan. 9, 1950.

January 1950 were California, Indiana, Maine, Maryland, Michigan, Montana, Nebraska, New Hampshire, New Jersey, Ohio, Oregon, South Dakota, Texas, Utah, and Washington.

c. Every plant health service and every plant, industrial unit, industry, and corporation should have a simple, workable, written plan to meet any foreseeable disaster. It should fit the means known to be available and every participant should know and understand his part.

d. We should initiate immediately all studies and projects which require attention locally or nationally. This should be done by preparing and publishing a list of all matters related to industrial health which would be involved in local or broader disaster control. The list should be arranged according to urgency or priority for action and rearranged regularly as indicated by experience.

e. We should consider everything possible to learn about the proper values and priorities to be assessed to various factors related to disaster control.

TABLE 2.—*Relationships in medical planning for disaster control*

Government agencies		*Civilian agencies*	
Civil:	Coordination, integration, and implementation	Plant	Management, Labor, Medical staff
Local			
State			
Military:		Industry	Management, Labor, Medical staff
Army Area Headquarters			
Naval District Headquarters			
Air Force Area Headquarters		Corporation	Management, Labor, Medical staff
National Security Resources Board	Advice and guidance	Plant, Industry, Corporation	

2. For long-range application of the lessons learned we should: (a) immediately insure a system to continue, revise, and maintain all plans (table 2); (b) continue to improve operational systems which have been designed in the interim, have been tested, and have been maintained for meeting disasters in a usable, stand-by status; (c) continue to improve those studies and research projects we shall initiate in the interim, adding to or deleting from them as required; (d) at the earliest practicable time determine the minimal quantity and quality of health services which would be required for industrial workers subjected to disaster, with particular attention to on-duty and off-duty requirements for the personnel themselves, as well as requirements for members of the community not employed by the industry, but whose health must be maintained to insure maximum productivity of the employees; (e) insure coordinated and judicious suggestions on all subjects or problems requiring investigation or research and which pertain to any field of industrial health concerned

with disaster control, and transmit them to the Research and Development Board, Department of Defense, if primarily military in nature, or to the National Research Council if not primarily military in nature but important to national defense; (f) actively sponsor the earliest practicable establishment of an authoritative and scientific classification of the entire population into categories of physical and mental fitness for specified duties or activities because only by this means can rapid, successful assignments to duties be made and large numbers of people shifted in emergencies to perform essential work in a safe and healthful way; (g) sponsor in a similar manner authoritative and scientific determination and publication of true minimal nutrient requirements for the predetermined categories of the whole population, for short- and long-term periods of survival or existence under various physiologic conditions, because only by such means will public food planning be undertaken scientifically, or can people subjected to major disasters be assured of adequate food;[30] (h) solve the problems related to fatigue, particularly its cause, prevention, and treatment as well as scientific measurement before, during, and after occurrence; (i) develop all the knowledge and technics necessary for maximum employment of women, the disabled or handicapped, the aged, persons of both sexes under 18 years old, employees shifted because of dispersal activities, persons involved in evacuations or migrations, and persons compelled to do productive work in any unpleasant, unhealthful, or hazardous environment, along with means and methods for maintaining or improving their health while employed; (j) develop the most efficient and economical administration of health services to be employed in disaster control; (k) learn the psychologic effects of and methods for handling inadequate nutrition, disasters, stresses of war, fatigue, or industrial environmental stresses; (l) apply all the known preventive and therapeutic measures to development of physical and mental health in the leaders and supervisors of industry and continue to seek new ones; (m) develop and train the best possible professional health leadership for industrial health services, particularly increasing their experience in sociologic factors; (n) develop all proper health means and measures which will insure placement of the proper employees in the proper duties, including the managing or supervisory staff, the employees, and the industrial health services' staffs; and (o) understand safety and accident prevention measures with particular attention to the cause, medical and administrative handling, psychologic aspects, and proneness.

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[30] The Food and Nutrition Board, National Research Council: Proceedings, Vol. IX, pp. 61–62, The National Research Council, Washington, D. C., Nov. 1949.

We could not improve on the theme furnished us many centuries ago by the famed Sun Tzu [31] when he said: "If the campaign is protracted, the resources of the State will not be equal to the strain" and "when your weapons are dulled, your ardour damped, your strength exhausted and your treasure spent, other chieftains will spring up to take advantage of your extremity. Then no man, however wise, will be able to avert the consequences that must ensue."

[31] See footnote 3, table 1.

Dentistry in the British Army

H. J. Higgins, *Major General, Royal Army Dental Corps*[1]
S. H. Woods, *Colonel, Royal Army Dental Corps*[2]

UP TO the outbreak of the South African War in 1899 dental treatment in the British Army may be said to have been non-existant. Although a dental pouch consisting of eight instruments, designed for the extraction and scaling of teeth, was authorized for the use of Army surgeons, the only interest taken in the oral condition of the soldier was to ensure that he possessed sufficient incisor teeth to enable him to bite the cap of the charger before passing the powder into the muzzle of his musket. In 1901 four civilian dental surgeons were dispatched to South Africa to treat troops in the field. In 1910 the employment of eight civilian dental surgeons in the United Kingdom and three in India, was approved.

At the outbreak of World War I the provision of dental treatment, for the Army, was still almost negligible and no dental surgeon accompanied the British Expeditionary Force to France in August 1914. Shortly after, however, dental surgeons were for the first time appointed to temporary commissions and attached to the Royal Army Medical Corps for duty, at home and overseas. At the time of the Armistice in 1918, a total of 850 dental officers were serving.

On 4 January 1921 the Army Dental Corps was formed as an integral part of the Army Medical Services and an Inspector, Army Dental Service, was appointed to the staff of the Director-General, Army Medical Services, at the War Office. This Corps was a joint service for the Army and the Royal Air Force until the formation in 1930, of the Royal Air Force Dental Branch.

In 1946 His Majesty the King graciously approved that the Army Dental Corps should be designated "The Royal Army Dental Corps" (RADC) and a new badge to replace the former one was also author-

[1] Director of British Army Dental Service.
[2] Consulting Dental Surgeon to the Royal Army.

ized. This new badge consists of "Within a laurel wreath, a dragon's head and sword; beneath a scroll bearing the motto 'Ex dentibus ensis'. The whole surmounted by a crown. The dragon's head and blade of the sword is in silver plate, the remainder of the badge—gilt."

Origin of the design.—The dragon is an emblem of dentistry in China, and is also associated with armies and teeth in the legends of Cadmus and Jason in Greek mythology. Both these characters are reputed to have slain dragons, sown their teeth, and reaped armies. The sword is intended to denote the branch of the Armed Forces to which the Corps belongs.

Administration of the RADC.—In addition to a Director and Assistant Director of Dental Service on the staff of the Medical Directorate at the War Office, Deputy Directors of Dental Service are borne on the establishment of the Medical Branch at Headquarters of Commands in the United Kingdom and overseas.

Specialist dental officers.—Dental officers who, by virtue of their experience, qualifications, et cetera, are classified as specialists are attached to the larger military hospitals, and, on active service, to maxillofacial teams.

Dental officers.—In addition to those serving with military hospitals, officers are assigned to dental centers, dental laboratories, mobile dental units, and under active service conditions to Casualty Clearing Stations [3] and Field Ambulances.[3] Dental centers vary in size from 1 to 20 officers, equipped with chairs according to requirements and are located in military camps, training establishments, and depots.

Commissions.—There are, at present, three types of commissions for dental officers (1) regular (permanent); (2) short service (for 4 years); and (3) National Service (for 18 months). Promotion to captain occurs after 1 year's service, to major after 8 years' service, to lieutenant colonel and above is by selection to complete establishment.

Training.—All newly commissioned officers on first appointment and enlisted personnel (dental technicians and dental operating room assistants) are assigned to the Depot and Training Establishment, RADC, for initial military training and instruction. In addition refresher and promotion courses are held as required. Selected officers are, from time to time, attached to civil plastic and head injuries hospitals.

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[3] See Armed Forces Medical Journal, Nov. 1950. pp. 1347 and 1349.

The following chronologic data indicate the main developments from 1660 to 1950

TABLE 1.—*The dental standards for recruits*

1660–1695	Musketeers required sufficient incisors to open the bandoleer (powder charge).
1678–1810	Grenadiers needed sufficient incisors to open the fuse of the grenade.
1695–1865	Whole of Infantry required sufficient incisors and canines to tear open the cartridge (combining powder charge and bullet).
1798	First instructions for medical inspection of recruits.
1821–1865	Loss of many teeth, particularly of the incisors and canines, was a cause for rejection.
1865	The modern pin-firing mechanism was introduced superseding the earlier cartridge, and incisors and canines lost their earlier significance.
1865–1898	Loss of many teeth was cause for rejection.
1899	"Recruits must possess a sufficient number of sound teeth for efficient mastication."
1906–1914	"Loss or decay of teeth to such an extent as to interfere materially with efficient mastication" was cause for rejection.
1921–1936	"The eleven-point standard," a simple, practical guide— Sound or repairable functional—Incisors, canines and premolars counted as one point each. First and second molars as two points each. Third molars (according to development) as one or two points each. Maximum possible points, 22 Minimum points required, 11; that is, 50 percent masticating efficiency.
1937	The standard was modified for other than front-line troops.
1939–1950	All standards in abeyance, except for candidates for commissions.

TABLE 2.—*Authorized dental equipment*

1660–1798	Army surgeon supplied his own.
1798–1820	1 key instrument.[1]
1820–1838	1 key instrument. 1 tooth forceps. 1 tooth lever.
1838–1857	1 key instrument. 2 tooth forceps. 1 punch. 1 gum lancet.
1857–1899	12 forceps. 6 elevators (to fit one handle). 1 key instrument. 1 gum lancet.
1900	8 forceps. 6 elevators. 1 mouth mirror. 1 gum lancet.
1904–1914	Restricted—modern.
1915–1950	Full scale—modern.

[1] Forerunner of elevator.

TABLE 3.—*Dental treatment, 1660–1950*

1660–1900	Extractions only, by Army surgeons.
South African War (1899–1902)	
1901–1902	General treatment (except dentures and repairs) by 4 contract dentists in the field.
1903–1908	Conservative treatment by 8 full-time contract dentists.
1909–1914	Limited conservative treatment by part time civilian contract dentists
World War I (1914–1918)	
1914–1921	All necessary treatment by temporary Army dental officers.
1921–1946	All necessary treatment by "The Army Dental Corps"—the first regular dental officers.
1947	Designation "Royal Army Dental Corps" and continuing full-scale treatment to date.

TABLE 4.—*Army dental officers, 1914–18*

1914	August–October.	None with	Expeditionary Force in France.
	November	12	For France only.
	December	20	For France only.
1915	February	36	(Including the first for Home.)
	August	150	
1916	August	300	(Compulsory Service Act.)
	December	460	
1917	December	500	
1918	May		First appointment of a dental officer to War Office to advise the Director-General, Army Medical Services, on dental matters.
	August	700	
	November	850	

TABLE 5.—*The Army Dental Corps, 1921–46*

		Officers	Other ranks
1921	Joint Service for Army and Royal Air Force	107	132
1930	(R. A. F. section separated to form R. A. F. dental service)	124	
1935		150	
1938		162	210
1939 (August)		237	325
1940 to 1945		2,300	3,500

TABLE 6.—*Royal Army Dental Corps, 1950*

Officers (Regular):	
Director, Army Dental Service (major-general) at War Office	1
Assistant Director, Army Dental Service (colonel)	1
Consulting Dental Surgeon to the Army (colonel)	1
Deputy Directors, Dental Service (colonels)	9
Commandant, Depot and Training Establishment, Aldershot (colonel)	1
Lieutenant-colonels	35
Majors, captains, and lieutenants as required to provide a ratio of 1 R. A. D. C. officer to 1,150 commissioned and enlisted personnel.	
Other ranks, R. A. D. C.:	
Approximately 1 dental operating room assistant to each R. A. D. C. officer.	
Approximately 1 dental technician to 3 R. A. D. C. officers.	

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The Use of Base Shops for Manufacture and Repair of Medical Appliances

BRUCE R. HEINZEN, *Captain, U. S. A. F. R. (MC)*[1]

THIS article is written to point out the possibility of effective cooperation between the medical department and the base shops of Air Force installations. Undoubtedly, installations in all service branches have used local facilities to aid in the care and treatment of medical and surgical patients. This is ordinarily sporadic. A close liaison, however, allows for an enhanced use of local facilities as well as a significant saving of money. Such appliances as Taylor type back braces (fig. 1), neck braces (figs. 2 and 3), and other items of this type can be constructed rapidly and at minimum cost by using salvaged pieces of equipment. Loss of time involved in transporting patients can thereby be avoided. This has been of increasing value since we have been using the Air Force convalescent rehabilitation program. Under this system, many man-hours of useful employment are obtained while a patient is in the late stages of recovery; and little or no time is lost from arranged hours of duty while a patient is undergoing fittings for an appliance.

Among the concomitant benefits from this liaison with personnel of the base shops is the noticeable feeling of teamwork which develops among the involved persons. In addition, the personalized attitude of the men making appliances invariably results in the achievement of small refinements in an appliance which increase the patient's comfort. In an organization such as a bomb wing, several activities in nonmedical organizations readily become applicable to the needs of the medical group. For example, the electronics shop will often be able to make minor repairs on cardiographs and physiotherapy equipment, reducing the number of calls for a medical maintenance man.

[1] Air Force Hospital, Chatham Air Force Base, Savannah, Ga.

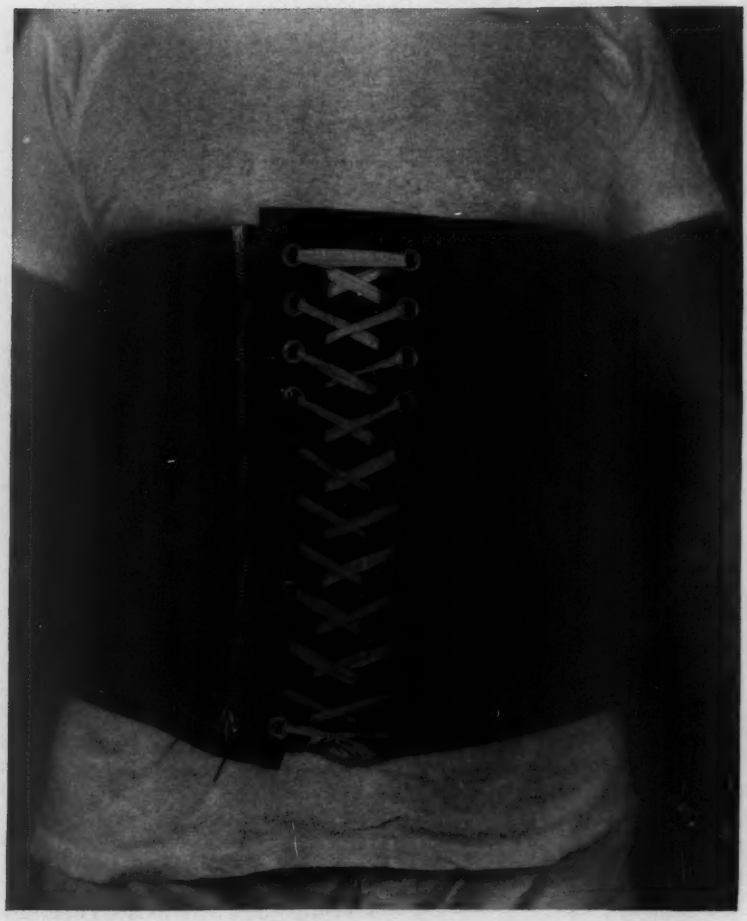

Figure 1.—Front view of base-constructed back brace used by patient with acute disk injury.

Figure 2.—Neck brace constructed at base shop used in treatment of dislocated cervical vertebra.

Figure 3.—Standard type commercial neck brace shown above and neck brace constructed at base shop shown below.

The sheet metal shops can rapidly repair such items as a broken cast-cutter, or can speedily construct effective walking irons if demands exceed supply levels unpredictably. Parachute shops can produce leather padding for metal appliances. The only payment ordinarily needed is recognition of a man's efforts and honest attempts to aid him with his medical problems.

About the Army Medical Service

Procurement of Professional Officers

PAUL I. ROBINSON, *Brigadier General, MC, U. S. A.*[1]

A REVIEW of the history of medical professional officer procurement for the past half century reveals an encouraging and continuing interest in professional training. The Army takes pride in its achievements over the years in the fields of preventive and curative medicine. These achievements need not be enumerated here because they are common knowledge to all medical officers. The advances in training fields have not so generally been made known. The fact is, however, that on each occasion when the Army has used training programs as a means of procurement, the response has been phenomenal. In 1926, the Regular Army Medical Corps was brought to strength by the introduction of Army internships. In this same period the Army Nurse Corps was greatly augmented by specialized training of young women in various fields of nursing. The Army has also been a leader in fostering dental postgraduate training in the form of internships and residencies.

After World War II, the deficits in various professional corps warranted expansion of training programs to an extent never before considered. All are acquainted with the military internship and residency programs and the civilian internship and residency programs for the Medical Corps; the Senior Dental Student and Dental Internship and Residency Programs for the Dental Corps; and the specialty training courses for nurses. Since V-J day, these programs have been instrumental in obtaining 1,296 officers for the Regular Army Medical Corps; 384 officers for the Regular Army Dental Corps; and about 500 officers for the Regular Army Nurse Corps.

Professional training in the Army Medical Service is now an established fact. There is no reason why professional officers in the Army should not be able to reach the pinnacle of their ambitions in professional achievements. They can participate on an equal basis with

[1] Personnel Division, Office of the Surgeon General, Department of the Army.

their contemporaries in civil life. What, exactly, have these training programs done to assist in accomplishing this procurement record? They have brought young professional people from civil life face to face with medical service in the Army and many have found that they like it. Since World War II, 311 medical interns entered the military program, exclusive of the present group, and 235, or 75.5 percent, have chosen to come in the Regular Army or remain on active duty for an extended period without obligation or compulsion.

In the immediate future, hundreds of young physicians, dentists, veterinarians, nurses, and allied medical scientists will be on duty with the Army as volunteers or through selective service. They must all, also, be made aware of the fine features of a medical career in the Army in order that they too may enter that career if they so choose. Each Regular Army officer must make himself available in official and social hours to discuss opportunities in the service with these young officers. They must understand that an attractive career awaits those who can qualify. Each Regular Army officer should consider himself a procurement officer in the sense that he must demonstrate by word and deed the advantages of an Army medical career.

The Army will continue, over the next several years, its programs for recruitment. At the end of from 4 to 6 years such procurement should be reduced to that of filling the vacancies of normal attrition. Dissatisfaction, in the meantime, can be held to a minimum if all officers on duty are kept busy and if they are kept informed. It is incumbent on every Regular Army officer to lend his aid to the accomplishment of these missions, to the end that the Medical Service will ever be enhanced to the glory and satisfaction of its members.

Recently Released Medical Films

The films, here listed, on medical and allied scientific subjects were recently released by the Medical Illustration Service, Armed Forces Institute of Pathology. These films, together with those previously released, are available on loan to members of the medical profession and allied scientific groups. Requests for the loan of films will be directed to the surgeon of the Army area in which the requesting institution or person is located: (1) Surgeon, First Army, Governors Island, N. Y., for those residing in Maine, Vermont, New Hampshire, Massachusetts, Connecticut, Rhode Island, New York, and New Jersey. (2) Surgeon, Second Army, Fort Meade, Md., for those residing in Delaware, Ohio, Pennsylvania, Virginia, West Virginia, Maryland, and Kentucky. (3) Surgeon, Third Army, Atlanta 3, Ga., for those residing in Tennessee, North Carolina, South Carolina, Georgia, Florida, Alabama, and Mississippi. (4) Surgeon, Fourth Army, Fort Sam Houston, Tex., for those residing in Texas, New Mexico, Oklahoma, Arkansas, and Louisiana. (5) Surgeon, Fifth Army, 1660 East Hyde Park Boulevard, Chicago, Ill., for those residing in Illinois, Indiana, North Dakota, South Dakota, Minnesota, Iowa, Wisconsin, Michigan, Wyoming, Nebraska, Colorado, Kansas, and Missouri. (6) Surgeon, Sixth Army, Presidio of San Francisco, Calif., for those residing in California, Washington, Montana, Idaho, Oregon, Nevada, Utah, and Arizona.

Number [1]	*Title*	*Running time (min.)*
PMF 5054 [2]	Therapeutic Exercise—Tendon Transplants	7
PMF 5055 [2]	Toward Independence (Paraplegic Rehabilitation)	30
PMF 5058	The Medical Effects of the Atomic Bomb—Pt. I. Physics—Physical Destruction—Casualty Effects.	32
PMF 5064	Vagotomy for Duodenal Ulcer	11
PMF 5065	Management and Technic of Pulmonary Lobectomy	27
PMF 5066	Electrodiagnostic Procedure	19
PMF 5070	Simple Anterior Closure of Colostomy	10
PMF 5073	Construction of Screw Lock Sectional Splint	17
PMF 5074	Types of Screw Lock Splints and Bone Graft Cases	13
PMF 5075	Reconstruction of the Lower Lip and Chin	18

See footnotes at end of table.

Number [1]	Title	Running time (min.)
PMF 5099	Neurolysis of the Ulnar Nerve in Lower Arm	8
PMF 5103	Arterial Disorders in the Upper Extremity and Their Treatment by Sympathectomy.	31
PMF 5105	Logistics In Reverse	13
PMF 5110	Radioactivity	17
PMF 5111	Demonstration of Cushing's Experiment on the Dog	14
PMF 5114	Rickettsiae—Laboratory Procedure for Their Isolation and Identification.	47
PMF 5116A [2]	Time Out: Occupational Therapy in Tuberculosis	27
PMF 5116B [2]	Occupational Therapy in Problems of Motion	24
PMF 5116C [2]	Journey To Reality (Occupational Therapy for Acute Psychotics).	40
PMF 5117	Tibial Nerve Anastomosis in the Lower Calf	7
PMF 5118	Diagnosis of Peripheral Nerve Injuries	16
PMF 5132	Method of Repair of Posterior Tibial Nerve	10
PMF 5133	Ulnar Nerve and Soft Tissue Defect and Simultaneous Repair in the Forearm.	10
PMF 5136	Benign Dental Tumors	29
PMF 5137	Malignant Oral Tumors	40
PMF 5140	Hereditary Ataxia	33
PMF 5143 [2]	Atomic Medical Cases—Japan, World War II	37
PMF 5148	The Medical Effects of the Atomic Bomb—Pt. II. Pathology and the Clinical Problem.	37
PMF 5149	The Medical Effects of the Atomic Bomb—Pt. III. Medical Service in Atomic Disaster.	28
PMF 5151 [3]	General Adaptation Syndrome	84
PMF 5152F	Psychiatric Interview Technique Part VI	
PMF 5152H	Psychiatric Interview Technique Part VIII	
PMF 5162 [2]	Operation of the Valves of an Ox Heart	4
PMF 5165	Typhoid Vaccine Research	29
PMF 5166 [3]	Electrocardiography	10
PMF 5169	Experimental Thiamin Deficiency	7
PMF 5170	Heart-Lung Preparation	17
	Miscellaneous Films	
MF 1396 [2]	Crossroads, Radiological Safety Motion Picture	25
	Training Films	
TF 8-1520 [2]	Isolation Technique	18
TF 8-1536 [2]	The Medical Examination, Part I, Importance	19
TF 8-1537 [2]	The Medical Examination—Pt. II, History	17
TF 8-1538 [2]	The Medical Examination—Pt. III, Examination	17
TF 8-1566 [2]	Veterinary Preventive Medicine	17
TF 8-1575 [2]	Hospital Food Service Personnel Training Introduction—Part I.	17
TF 8-1576 [2]	Hospital Food Service Personnel Training—Part II. Personal Appearance, Hygiene and Sanitary Food Handling.	33
TF 8-1577 [2]	Hospital Food Service Personnel Training—Part III. Equipment.	13

See footnotes at end of table.

Number [1]	Title	Running time (min.)
TF 8-1586	Fractures—An Introduction	27
TF 8-1639 [2]	Medical Supply—Armed Forces	62
TF 8-1670 [2]	Habits and Characteristics of the Rat—Pt. I. The Norway Rat.	29
TF 8-1671 [2]	Habits and Characteristics of the Rat—Pt. II. The Roof Rat.	13
TF 8-1571 [2]	First Aid—Part I. Major Wounds, Fractures and Burns.	38
	Film Bulletins	
FB 223 [2]	Veterinary Service With Army Animals—Part II. Evacuation and Treatment of Animal Casualties.	24
FB 225 [2]	Veterinary Service With Army Animals—Part III. Injuries and Diseases of Army Animals, World War II.	30
FB 228 [2]	Veterinary Food Inspection Service—Part II. Theater of Operations.	16

[1] All films have sound track.

[2] Black and white.

[3] Limited distribution. Request must be sent direct to Director, Armed Forces Institute of Pathology; Attention: Medical Illustration Service.

Public Health Aspects of Biologic Warfare[1]

Biologic warfare is the use of disease agents, or their toxic products, to produce disease or death in man, animals, or crops. This is public health in reverse. This method of warfare is not to be casually dismissed, nor should it strike terror in our minds. Many exaggerated and sensational statements, such as the widely quoted statement that 1 ounce of botulinus toxin can kill 200,000,000 people, have appeared in the press. Such quantities of materials could not be disseminated widely enough by an enemy to produce illness or death among more than a small fraction of such fantastic numbers of persons. The potentialities of biologic warfare are great, but in its present stage of development it should not be considered a means of mass destruction comparable to atomic warfare. Organisms that have been notorious producers of accidental infections among laboratory workers are the types of agent most likely to be used. The attempt could be made to introduce diseases which are already of usual, or endemic, occurrence in the area under attack because such an outbreak might not be recognized as warfare at all. The mere occurrence of an unusual disease, on the other hand, should not be labeled biologic warfare unless there is other evidence to substantiate such a claim.

It is possible to select agents which in effective doses could result in (1) a high fatality rate, (2) prolonged incapacitation with low fatality, or (3) only temporary illnesses. Although there are potential agents which spread rapidly from person to person, it is not at all certain that such a spreading epidemic could be set up at will. Even if an agent capable of direct transmission from person to person were used, public health and sanitary measures could limit the outbreak and minimize its effects, especially if coupled with adequate prophylactic and therapeutic agents.

If biologic warfare were initiated, it might be launched by the use of missiles and munitions, capable of transport by aircraft, and designed to set up airborne clouds of biologic agents. Such use would be similar to chemical warfare, except that the effects would be delayed because of the incubation periods of the agents. Such munitions might be used in conjunction with blast weapons in order to take advantage

[1] ABSTRACT: PHILBROOK, FRANK R. (Commander, MC, U. S. N.): Consideration of certain public health aspects of defense against biological warfare. Read at the American Public Health Association Meeting, St. Louis, Mo., 31 October 1950.

of disrupted sanitation and medical service. The aerosols of biologic warfare agents produced under such conditions would rapidly disperse into infective clouds which would be odorless, tasteless, and invisible and, thus, extremely difficult to detect. An important defense is military interception and prevention of the attack. Another method of dispersal involves the clandestine introduction of those agents into the air, food, or water supplies by saboteurs. Internal security measures directed against the saboteur or against his access to food, water supplies, or important areas are the important defensive measures.

The greatest return for the least expended effort would be the use of biologic warfare in thickly populated areas. The greatest defensive effort must be directed toward these likely target areas and others such as key industrial, communication, and governmental centers. Every competent epidemiologist, physician, bacteriologist, veterinarian, nurse, or sanitarian who is well-informed regarding the communicable diseases has already acquired the information necessary to the formulation of many civil defense principles. A few such principles are: (1) Civil defense against biologic warfare must be coordinated with defense against other forms of warfare. (2) Biologic warfare agents produce the usual disease characteristics of the given agent, although the clinical course may be expected to vary. (3) Management and control of communicable diseases, including isolation and quarantine, remain the same whether the diseases occur in the natural course of events or are introduced by an enemy. (4) Preparedness for rapid diagnosis and therapy is important not only for those diseases of usual, or endemic, occurrence but also for those considered exotic or of unlikely occurrence except by artificial dissemination. (5) Plans for availability of chemotherapeutic and antibiotic substances should be made. (6) Immunization might be presumed to afford a measure of specific protection against potential agents. (7) Gas masks afford considerable protection and would be of value if an adequate warning of probable biologic or chemical attack could be given. (8) Food and drinking water, if suspected of contamination by biologic warfare agents, may be sufficiently sterilized by boiling. (9) Public health authorities must be prepared to cope with the panic which any unusual incidence of disease is apt to cause. (10) The speed of modern transportation, considered together with the incubation period of disease caused by potential agents capable of person-to-person spread, is important as a factor in biologic warfare. (11) Specialized training involving access to the more highly classified biologic warfare information for a few well-qualified epidemiologists and public health personnel in key State, territorial, or local positions is desirable. (12) The task of planning civil defense has been charged to the National Security Resources Board.

Navy's Color Atlas of Pathology

The *Color Atlas of Pathology*, prepared by the Navy and the first such comprehensive work of its kind in the world, was published in November 1950 by the J. B. Lippincott Co. of Philadelphia, Pa. In process of preparation over a period of 6 years, the Atlas is looked upon as an important contribution to medicine and its allied sciences; it will serve as a useful tool in the study and interpretation of both gross and microscopic findings in pathology.

The Medical Department of the Navy has long recognized the need for a means of providing a more adequate background in pathology to a wider range of students of the medical sciences. It is difficult for teaching institutions always to secure a sufficiently large representation of specimens for study; and practicing physicians have often lamented the lack of access to files of material for diagnostic work of their own. Only in recent years, with the advent of new processes for accurate color photography and printing, has it been within the realm of possibility to assemble and publish a guide designed to meet these deficiencies.

The ground work for this *Color Atlas of Pathology* was laid by the U. S. Naval Medical School of the National Naval Medical Center at Bethesda, Md., in 1944, and numerous obstacles had to be overcome in the succeeding years finally to bring this endeavor to fruition.

A number of the requisites essential to such an undertaking were concomitants of World War II. Chief among these should be mentioned the availability of artists of particular talent and training. Also important was the opportunity to use material belonging in the Pathology Department of the Navy's Medical School, the Army (now Armed Forces) Institute of Pathology, Johns Hopkins Hospital, and Georgetown University. These rich sources of material facilitated the correlation of clinical histories and findings with related pathology, adding immeasureably to the value of the presentations.

The Navy was most fortunate to find in the person of Commander Charles F. Geschickter, Medical Corps, U. S. Naval Reserve, wartime head of the Department of Pathology at the Naval Medical School, one who was eminently qualified to evaluate, correlate and to assimilate this material. Doctor Geschickter worked with Commander

W. W. Ayres, Medical Corps, U. S. Navy, and currently Chief Pathologist, U. S. Naval Medical School, under the direction of Rear Admiral Lamont Pugh, Medical Corps, U. S. Navy, currently Deputy Surgeon General of the U. S. Navy but who was commanding officer of the U. S. Naval Medical School when the Atlas project was in its incipiency and who has continuously been a leading figure in its progress and ultimate consummation.

The *Color Atlas of Pathology* is the first of three such volumes and covers hematology, spleen and thymus, lymph nodes and tonsils, the respiratory system, the liver, oral cavity, gastrointestinal tract, heart and blood vessels, kidney and urinary tract, and the skeletal system. The second volume dealing with pathologic conditions peculiar to the specialities (neuropathology, dermatology, endocrinology, et cetera), and a third volume devoted entirely to oral and dental pathology are in process of preparation. These two additional volumes are being compiled and edited under the auspices of the U. S. Naval Medical and U. S. Naval Dental Schools respectively at the National Naval Medical Center, Bethesda, Md. The volume devoted to pathology of the specialties will probably be ready for publication in 1953 or 1954. The volume on oral pathology will probably be published much earlier.

BOOKS RECEIVED

Oral Pathology, A Histological, Roentgenological, and Clinical Study of the Diseases of the Teeth, Jaws, and Mouth, by Kurt H. Thoma, D. M. D., F. D. S., R. C. S. (Eng.), F. D. S., R. C. S., h. c. (Edin.), *Professor of Oral Surgery, Emeritus, and Brackett Professor of Oral Pathology, Harvard University; Honorary Professor of the Odontologic Faculty, San Carlos University, Guatemala; Lecturer in Oral Surgery, Graduate School of Medicine, University of Pennsylvania; Member of Board of Consultation, Formerly Oral Surgeon and Chief of Dental Service, Massachusetts General Hospital; Oral Surgeon to Brooks Hospital; Consulting Oral Surgeon, New England Baptist Hospital, Beth Israel Hospital, Tumor Department of Boston Dispensary, New England Center Hospital, Faulkner Hospital; Consultant in Oral Surgery, Army Medical Center, and Consultant in Oral Pathology, Armed Forces Institute of Pathology, Washington, D. C.* 3d edition. 1,592 pages, with 1,660 illustrations, including 78 in color. The C. V. Mosby Company, St. Louis, Mo., publisher, 1950. Price $17.50.

Thromboembolic Conditions and Their Treatment With Anticoagulants, by Charles D. Marple, M. D., *Assistant Clinical Professor, Division of Medicine, University of California Medical School, San Francisco, Calif.; formerly Research Fellow, Department of Medicine, Cornell University Medical College, and Assistant Physician to Out-Patients, The New York Hospital, New York City,* and Irving S. Wright, M. D., *Professor of Clinical Medicine, Cornell University Medical College, and Attending Physician at The New York Hospital, New York City.* 418 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1950. Price $8.50.

Urgent Diagnosis Without Laboratory Aid, a Discussion of the External Signs of Conditions Which Threaten Life, by Prof. Dr. Hanns L. Baur, a.o. *Professor of Internal Medicine, University of Munich; formerly, Medical Director and Physician-in-chief, Hospital Munich-Schwabing.* Publication Number 66, American Lecture Series. 89 pages. Charles C Thomas, Publisher, Springfield, Ill., 1950. Price $2.

Neurosis and Psychosis, by Beulah Chamberlain Bosselman, M. D., *Associate Professor of Psychiatry, University of Illinois College of Medicine, Chicago, Ill.;* foreword by Francis J. Gerty, M. D., *Professor of Psychiatry and Head of the Department, University of Illinois College of Medicine, Chicago, Ill.* 172 pages. Charles C Thomas, Publisher, Springfield, Ill., 1950. Price $4.50.

A Manual of Physics, by J. A. Crowther, Sc. D., F. Inst. P., *Sometime Fellow of St. John's College, Cambridge: Professor Emeritus of Physics in the University of Reading.* 5th edition. 594 pages, illustrated. Oxford University Press, New York, N. Y., publisher, 1950. Price $4.25.

Researches in Binocular Vision, by Kenneth N. Ogle, Ph. D., *Section on Biophysics and Biophysical Research; Research Consultant in the Section on Ophthalmology, Mayo Foundation and Mayo Clinic, Rochester, Minn.* 545 pages, illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1950. Price $7.50.

An Integrated Practice of Medicine, Volume V, pages 4133–4867, by Harold Thomas Hyman. 734 pages. W. B. Saunders Co., Philadelphia, Pa., publisher, 1950. Price $10.

Bronchoesophagology, by Chevalier Jackson, M. D., Sc. D., LL. D., F. A. C. S., *Honorary Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia,* and Chevalier L. Jackson, M. D., M. Sc., F. A. C. S., *Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia.* 366 pages, illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1950. Price $12.50.

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The 1950 Year Book of Radiology (June 1949–June 1950). Radiologic Diagnosis edited by Fred Jenner Hodges, M. D., *Professor and Chairman, Department of Roentgenology, University of Michigan,* and John Floyd Holt, M. D., *Associate Professor, Department of Roentgenology, University of Michigan.* Radiation Therapy edited by Isadore Lampe, M. D., *Associate Professor, Department of Roentgenology, University of Michigan,* and Robert S. MacIntyre, M. D., *Assistant Professor, Department of Roentgenology, University of Michigan.* 480 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., publisher, 1950. Price $6.75.

Pediatric X-ray Diagnosis, A Textbook for Students and Practitioners of Pediatrics, Surgery and Radiology, by John Caffey, A. B., M. D., *Professor of Clinical Pediatrics, College of Physicians and Surgeons, Columbia University; Attending Pediatrician and Roentgenologist, Babies Hospital, and Vanderbilt Clinic, New York City; Consulting Pediatrician Grasslands Hospital, Westchester County, N. Y., and New Rochelle Hospital, New Rochelle, N. Y.; Consulting Roentgenologist, Orange Memorial Hospital, Orange, N. J.; Consultant in Pediatric Roentgenology, The New York Hospital, New York City.* 2d edition. 802 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., publisher, 1950. Price $22.50.

The Community and Public Health Nursing, A Handbook for and About Boards and Citizens Committees, by Edith Wensley, For The National Organization For Public Health Nursing. 250 pages. The Macmillan Co., New York, N. Y., publisher, 1950. Price $3.50.

Principles of General Psychopathology, An Interpretation of the Theoretical Foundations of Psychopathological Concepts, by Siegfried Fischer, M. D., *Clinical Instructor in Psychiatry, University of California, Formerly Professor of Psychiatry and Neurology, University of Breslau.* 327 pages; illustrated. Philosophical Library, New York, N. Y., publisher, 1950. Price $4.75.

Manson's Tropical Diseases, A Manual of the Diseases of Warm Climates, edited by Sir Philip H. Manson-Bahr, C. M. G., D. S. O., M. A., M. D., D. T. M. and H. Cantab., F. R. C. P. Lond., *Past President of the Royal Society of Tropical Medicine and Hygiene, London, and the Medical Society of London. Consulting Physician to the Hospital for Tropical Diseases, London; the Albert Dock Hospital and Tilbury Hospital. Consultant in Tropical Diseases to the Admiralty. Formerly Consulting Physician to the Colonial Office and Crown Agents for the Colonies. Formerly Consultant in Tropical Diseases to the Royal Air Force and Ministry of Pensions. Late Director, Division of Tropical Medicine, London School of Hygiene and Tropical Medicine; and Lecturer on Tropical Medicine to the London Hospital; Corresponding Member of the Société de Pathologie Exotique and of the Soc. Belge de Med. Trop. Member of the Washington Academy of Medicine; Examiner in Tropical Medicine to the Conjoint Board of the Royal College of Physicians and Royal College of Surgeons, England, Late Examiner to Cambridge and Hongkong Universities. Author (with A. Alcock) of "The Life and Work of Sir Patrick Manson," 1927; "The Dysenteric Disorders," 1939, and "Synopsis of Tropical Medicine," 1943.* 13th edition. 1,136 pages, with 17 colour plates, 9 half-tone plates, 412 figures in the text, 8 maps and 29 charts. The Williams & Wilkins Co., Baltimore, Md., publisher, 1950. Price $9.

Differential Diagnosis of Internal Diseases, Clinical Analysis and Synthesis of Symptoms and Signs, by Julius Bauer, M. D., F. A. C. P., *Clinical Professor of Medicine, College of Medical Evangelists, Los Angeles; Senior Attending Physician, Los Angeles County General Hospital; Consultant in Medicine, White Memorial Hospital, Long Beach Veterans' Administration Hospital, and Cedars of Lebanon Hospital, Los Angeles; Formerly Professor of Medicine, University of Vienna.* 866 pages; illustrated. Grune & Stratton, New York, N. Y., publishers, 1950. Price $12.

Evaluation in Physical Education, Better Teaching Through Testing, by M. Gladys Scott, *Professor of Physical Education, State University of Iowa,* and Esther French, *Professor and Head, Department of Health and Physical Education for Women, Illinois State Normal University.* 348 pages; illustrated. The C. V. Mosby Co. St. Louis, Mo., publisher, 1950. Price $4.

BOOK REVIEWS

Nursing in Prevention and Control of Tuberculosis, by H. W. Hetherington, M. D., M. R. C. P. (London), *Chief of Clinic of the Henry Phipps Institute of the University of Pennsylvania; Assistant Professor of Medicine of the University of Pennsylvania School of Medicine; Former Visiting Physician to the White Haven Sanatorium;* and Fannie W. Eshleman, R. N., B. S., *Supervisor of Public Health Nursing of the Henry Phipps Institute of the University of Pennsylvania; Lecturer on Tuberculosis Nursing, Department of Nursing Education of the University of Pennsylvania.* Revised 3d edition. 361 pages; illustrated. G. P. Putnam's Sons, New York, N. Y., publishers, 1950. Price $4.50.

This book is a summarization of the nursing of the tuberculous patient, including the health supervision of the patient's family, case finding, prevention and control of tuberculosis in communities and among personnel caring for tuberculous patients. There are many requirements for good care and understanding of the tuberculous patient. The nurse must be well-informed on the subject and have good insight into the physical, emotional, and mental needs of each patient. This text includes new material in many of these fields. The public health aspect, rehabilitation, and patient training are stressed. In addition, basic principles of medical and surgical nursing, aseptic technic, and a general history of the disease are discussed.

The chapter on diagnosis explains the importance of the x-ray and laboratory procedures that are most frequently used. Tuberculin testing and BCG vaccination are discussed. The chapter on prevention describes the accepted physical facilities and methods of sterilization used in the care of tuberculous patients, both in the hospital and in the home. Many of the recommendations for the physical facilities in hospitals would be an excellent guide for hospital administrators and nurses in promoting the safety and welfare of their patients and staff. Although this book would give the student a clear, concise idea of the nursing care, aseptic technic, and socioeconomic problems of the patient in the modern tuberculosis hospital, supplementary textbooks and references would also be necessary. To this end each chapter is followed by a list of such references.

—Lt. A. Baer, NC, U. S. N.

Primer of Allergy, a guidebook for those who must find their way through mazes of this strange and tantalizing state, by Warren T. Vaughan, M. S., M. D., *Richmond, Va.* 175 pages; illustrated. 3d edition, revised by J. Harvey Black, M. D., *Dallas, Tex.* The C. V. Mosby Co., St. Louis, Mo., publishers, 1950. Price $3.50.

This primer, originally written by one of our outstanding allergists, the late Dr. Vaughan, has been ably revised by Dr. Black. It does not go as deeply into the subject as a text would, but gives the essential features of allergy and its problems, especially for physicians in other fields. It is also a ready reference for any patient who would like to read up on the subject. It is well illustrated and contains an interesting series of questions and answers at the end.

—Lt. Col. A. J. Berger, MC, U. S. A.

Saints, Sinners and Psychiatry, by Camilla M. Anderson, M. D., *Assistant Clinical Professor of Psychiatry, University of Utah.* 206 pages. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1950. Price $2.95.

This monograph presents a theory of normal and abnormal behavior in terms of disturbances in the structure or function of the personality. The author describes her theory as "a new formulation of the dynamics of behavior which has some details in common with other theories (Freud, Adler, Sullivan, Schilder) but yet is distinctly different from all of them." Although one is inclined to question the need for any more psychiatric books intended for the layman because the market seems to be glutted with them, the author's lucid and nontechnical presentation makes for easy reading if the reader is seeking an explanation as to why we behave as we do. On the other hand, the nondiscriminating layman may be duped into believing that the author is as accomplished and learned in ethics, theology, and philosophy as she is in psychiatry. Actually, the ethical philosophy advanced is a combination of secularism and pragmatism, and implies that our behavior would be more "realistic" were we to discard moral values. The title is misleading in that we learn nothing of how saints or sinners operate since the author confines her remarks to people who merely think they are saints or sinners. The viewpoint expressed is a combination of psychobiologic concepts plus an essentially non-Freudian psychoanalytic orientation. The realistic appraisal and acceptance of parents' shortcomings strikes a wholesome note at a time when parents are being made the scapegoats for all children's troubles. Residents in psychiatry may well derive more from this book than from many a longer text on psychiatry or psychopathology. It has little to offer the experienced psychiatrist other than to acquaint him with current theories of behavior, the presentation of which were better made in scientific journals, rather than in a popular book with unfortunate digressions into the realms of ethics and metaphysics.—*Commander J. F. McMullin, MC, U. S. N.*

Malignant Disease and Its Treatment by Radium, by Sir Stanford Cade, K. B. E., F. R. C. S., M. R. C. P., *Surgeon, Westminster Hospital; Consulting Surgeon, Mount Vernon Hospital and Radium Institute; Lecturer in Surgery, Westminster Medical School and formerly Examiner in Surgery, University of London; Member of the Council and of the Court of Examiners, late Hunterian Professor and Arris and Gale Lecturer, Royal College of Surgeons of England; Hon. Member American Radium Society; Consultant in Surgery to the Royal Air Force,* with a foreword by Sir Ernest Rock Carling, F. R. C. P., F. R. C. S., F. F. R., *Consulting Surgeon and Vice-President, Westminster Hospital.* Volume III, 2d edition. 446 pages, illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1950. Price $12.50.

The author in his preface to the first edition stated that Volume III described the natural history and treatment of malignant diseases. In the preface to the present edition, he states that Volume III deals with the common neoplasms of the breast and thorax, the abdomen and pelvis, and the male and female external genitalia. In perusing the volume, one will search in vain to find a large group of malignant diseases within the abdomen even mentioned. The most conspicuous omissions are the malignant neoplasms of the small intestine, cecum, colon, and kidneys. This volume consists of 7 chapters and covers malignant diseases of the breast, esophagus, stomach, uterus, vagina, female urethra, vulva, ovary, bladder, penis, rectum, anus, and intrathoracic tumors. The author himself has written the chapter on the breast and a chapter on the bladder and penis. The other chapters have been written by or in collaboration with other British authorities.

The clinical types, natural history, symptoms and signs of the various neoplasms are well described. The subject of diagnosis is well fortified by excellent pathologic concepts. Prognosis is well discussed and soundly evaluated on the basis of wide knowledge and extensive experience. The author's philosophy with regard to using both surgical and radiologic treatment when indicated serves to emphasize that teamwork rather than competition between surgeons, radiologists, and pathologists is necessary if the greatest number of patients is to receive the best therapy. The author does not go into the details of operative technic but radiation therapy is discussed at length reflecting perhaps the preponderance of the author's own experience. The book's greatest value will be to the general practitioner and radiologist. The surgeon and pathologist engaged in tumor clinic work will also find the book useful.

—*Lt. Col. R. S. Aronson, MC, U. S. A.*

An Index of Tumor Chemotherapy, a tabulated compilation of data from the literature on clinical and experimental investigations, by Helen M. Dyer, *Biochemist, National Cancer Institute, National Institutes of Health.* Distribution to persons engaged in cancer research or some allied field, free of charge, by Federal Security Agency.

This large, paper-bound book has been prepared to furnish investigators in cancer research with a comprehensive survey of the literature on the treatment of tumors by chemical methods. In evaluating this index, the reader must remember that the Council on Pharmacy and Chemistry of the American Medical Association considers that the presently established treatment for cancer consists of the judicious use of surgical operations and/or irradiation. For years investigators have been trying to find a chemical substance which would destroy the cells of the invading tumor and spare the normal cells. Ehrlich was one of the pioneers in this field. Since his time hundreds of investigators have used different chemicals in this type of investigation. The reports of these studies are many and varied and scattered throughout the world's medical literature. Those investigating cancer will probably find this index valuable as they may look up the results of the use of various types of chemicals in the treatment of malignancy and, if interested, read of the work in the original articles which are listed. This compilation of data represents a tremendous amount of work. The average practicing physician and surgeon would not have much use for it but should know of its existence.—*Col. Dean M. Walker, MC, U. S. A.*

Therapeutic Radiology, by George Winslow Holmes, M. D., *Radiologist, Waldo County General Hospital, Maine; Honorary Physician, Massachusetts General Hospital; Radiologist-in-Chief, Massachusetts General Hospital 1916–1941; Clinical Professor of Roentgenology, Emeritus, Harvard Medical School,* and Milford D. Schulz, M. D., *Radiologist, Massachusetts General Hospital; Instructor in Radiology, Harvard Medical School.* 347 pages; with 121 illustrations, 10 in color. Lea & Febiger, Philadelphia, Pa., publishers, 1950. Price $7.50.

The authors state in the preface that their object is "to present in a concise manner the development, principles, and use of radiation, especially with the roentgen ray, in the treatment of disease." This they have done in their excellent introduction to roentgen radiation therapy. Mention of radium (except for cancer of the cervix) and radioisotopes is almost entirely limited to references. This is not a drawback, as it keeps the book to a reasonable size, and, after all, the fundamental radiation effect is essentially the same. Also, to avoid controversial discussions, it has occasionally been necessary to be rather brief and somewhat dogmatic, which is acceptable from a teaching point of view.

The table of contents is comprehensive. About one-third of the book is devoted to history, fundamental concepts of physics, biologic effects, characteristics of various sources of radiation, and tumor responses, followed by a few pages on the preparation and care of patients. The major portion of the book is anatomically presented. A most useful feature of this section for instructional purposes is the incorporation of typical case histories, which present the problem, how it was met, specific factors used for therapy, reactions obtained, and the follow-up. It is from these specific case studies that the illustrative material is selected. The illustrations are of good quality, and the diagrams are practical. The last two chapters cover radiation protection, with perhaps a little too much data on radium, and advice on medico-legal problems of therapeutic radiology.

—*Capt. Z. S. Gierlach, MC, A. U. S. (Inact.)*

Pathologic Physiology: Mechanisms of Disease, edited by William A. Sodeman, M. D., F. A. C. P., *The William Henderson Professor of the Prevention of Tropical and Semi-Tropical Diseases, Tulane University of Louisiana School of Medicine; Senior Visiting Physician, Charity Hospital of Louisiana; Consultant in Medicine, U. S. Marine Hospital at New Orleans.* 808 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publishers, 1950. Price $11.50.

This volume is a collaborative effort by 25 authors who are experts in their respective fields. The subject matter encompasses the circulatory, respiratory, and digestive systems; the blood and spleen, the urinary tract, the endocrine glands, water balance, nutrition, the locomotor system, infectious diseases, allergy, and physical, toxic, and chemical agents. Each of these subjects is discussed in the light of the most modern concept of disturbed physiology. This text encompasses all the fields of medicine with special emphasis on the problems of internal medicine. Nowhere is there a better compilation of this material than in this volume. It is of the utmost value for the student, the practicing physician, and the teacher of medicine.

—*Commander H. A. Lyons, MC, U. S. N.*

Endodontia, The Clinical Pathology and Treatment of the Dental Pulp and Pulpless Teeth, by Edgar D. Collidge, B. S., M. S., D. D. S., LL. D. (Hon. Loyola) *Emeritus Professor of Therapeutics, Preventive Dentistry and Oral Hygiene, Chicago College of Dental Surgery, School of Dentistry, Loyola University, Chicago, Ill.; formerly Professor of Materia Medica, Pharmacology and Therapeutics of the School of Dentistry, University of Illinois.* 300 pages; 355 illustrations on 179 figures and a colored plate. Lea & Febiger, Philadelphia, Pa., publishers, 1950. Price $6.

The author's knowledge and practical application of therapeutic principles is made evident early in the text when he discusses such topics as the pharmacologic action of drugs, immunity to disease, and etiologic and symptomatic treatment of disease. In greater detail the author discusses the action of arsenic in pulp devitalization and of paraformaldehyde in pulp mummification. He gives a good description of the pharmacologic action and therapeutic values of chloramine solutions in disinfecting root canals and of Lugol's solution as a root canal dressing to stimulate repair of disturbed periapical tissues. Of great value is his discussion of the use of sulfonamides and other antibiotics in the treatment of infected canals. He stresses the importance of being familiar with the pharmacologic action of the various drugs and remedies used in preparing the patient for operation and in the control of pulpal diseases. The technic for treating, preparing, and filling of root canals of pulpless teeth is well covered

with many fine illustrations to supplement the text. The indications and technic for root resection are thoroughly explained.

The most valuable sections of the book are those in which the author demonstrates the futility of the concept that once the pulp of a tooth has been removed or destroyed, that tooth can no longer be retained as a living and functioning member of the dental arch. By means of numerous roentgenograms and histologic sections the author shows how healing takes place in and about the periapical rarefactions once the source of irritation in the canals is properly eliminated, i. e., by complete disinfection and obliteration of the canal. The author stresses the idea that the root filling must completely obliterate the root canal in length and diameter in order to avoid accumulation and stagnation of lymph and tissue exudate which interferes with or completely checks the process of repair.

A detailed description of various pulpal and periapical disturbances from pulpal hyperemia to pulp necrosis and gangrene through granulomas and cysts with their various clinical manifestations by both subjective and objective signs and symptoms plus complete correlative radiographic and histologic illustrations of the pulpal lesions found in each condition is an invaluable aid in making a diagnosis of the state of the pulp on first presentation. It also gives to the operator a clear picture of the conditions he has to contend with and serves as a guide in the treatment to be used.

In this work the rationale behind present-day methods of retaining pulpless teeth is so clearly and forcefully presented that no one in the practice of the healing arts can have any qualms or reservations as to the fundamental scientific correctness of this practice. This book should prove extremely valuable as a text for students and as a handy reference work to the dentist in active practice.—*Capt. M. Dicher, U. S. A. F. (DC)*

The Meaning and Practice of Psychotherapy, by V. E. Fisher, Ph. D., *Psychologist and Psychotherapist. Formerly Assistant Psychologist, Worcester State Hospital; Assistant Professor of Psychology and Director of the Mental Clinic, New York University, Washington Square College; Psychologist and Psychotherapist, Idaho State Hospital South.* 411 pages. The Macmillan Co., New York, N. Y., publishers, 1950. Price $5.

The expressed purpose of the author of this book is to describe and illustrate such procedures and technics as he has found to be most effective. It is addressed to advanced students of psychology in the abnormal, clinical, therapeutic, and counseling fields; to psychotherapists, psychiatrists, psychologic counselors, and social workers. The author gives a definition of psychotherapy and lists various psychotherapeutic technics. The book is divided into four parts. Part 1, "A General Orientation," includes a suggested method of history taking, a description of the use and value of psychologic testing, and suggests and describes the author's method of management of the patient. Part 2, "Some Psychotic and Closely Related Disorders," includes early schizophrenic and other psychotic disorders considered by the author to be amenable to psychotherapy, psychopathic, and hypochondriacal reactions. In Part 3, "Psychoneurotic Reactions," the author discusses anxiety equivalent reactions, anxiety hysteria, phobias, and mental regression. He justifies the use of the term anxiety equivalent by stating it is more correct than the popular term, psychosomatic. Part 4, "Some Maladjusted Psychosocial Tendencies and Reactions," includes a brief evaluation of suicidal tendencies, compulsive drinking, feelings of inferiority, homosexual tendencies, psychic impotence, sexual frigidity, and marital discord. The author uses case histories to illustrate his points.—*Commander H. S. Colony, MC, U. S. N.*

Cytology of the Human Vagina, by Inés L. C. De Allende, M. D., *Chief of the Division of Endocrinology, Mercedes and Martin Ferreyra Institute of Medical Investigation,* and Oscar Orías, M. D., *Director, Mercedes and Martin Ferreyra Institute of Medical Investigation, Córdoba, Argentina,* with a foreword by Bernardo A. Houssay, M. D., translated from the Spanish by George W. Corner, M. D. 286 pages. Paul B. Hoeber, Inc., New York, N. Y., publisher, 1950. Price $7.50.

In this book the authors attempt to correlate some of the cytologic findings in vaginal secretions with the various phases of the menstrual cycle, normal and abnormal, with clinical endocrinopathies and to provide a basis for estimating the optimal hormone treatment. The approach is not new, but is a continuation of Shorr's work. A great deal of original work has gone into the preparation of the book. It opens new vistas for research in functionally normal and abnormal conditions particularly as they may be manifested in the epithelium of the vagina and cervix. The authors have made a beginning in accumulating data which may lead to a clinical method of evaluating the need for supplementary hormones, selecting the one or ones required, and interpreting the results obtained. The translator is to be commended for his lucidity. The book is exceptionally well illustrated. Graphs and tables are effectively used.

—*Commander Roy E. Crowder, MC, U. S. N.*

The 1950 Year Book of Medicine (May 1949–May 1950). Edited by Paul B. Beeson, M. D., J. Burns Amberson, M. D., William B. Castle, M. D., S. M. (Hon.) Yale, M. D. (Hon.) Utrecht, Tinsley R. Harrison, M. D., and George B. Eusterman, M. D. 819 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., publishers, 1950. Price $5.

This book reviews current medical literature from May 1949 to May 1950, under the following headings: Infections; The Chest; Blood and Blood Forming Organs; Heart, Blood Vessels and Kidneys; and The Digestive System. In addition to the usual succulent editorial comments each editor has prepared an introductory chapter reviewing the significant medical advances during the decade 1940–50. This is exciting reading and readily impresses one that medicine today is far from static. The range of the subject-matter and the extensiveness of the literature covered in this book is such that no practitioner has the time or background to review and appraise the significant contributions in all branches of internal medicine. The several editors together have attempted to cover the field and therefore this is required reading for the astute internist or general clinician. The only criticism offered is that in consideration of BCG vaccine attention was directed toward its drawbacks and our ignorance concerning its mode of action without mention of the increasing evidence that vaccinated persons have a significantly decreased morbidity and mortality from tuberculosis and that vaccination is a relatively innocuous procedure.

—*Lt. Col. W. J. Walker, MC, U. S. A.*

Medical Parasitology, for Medical Students and Practicing Physicians, by William G. Sawitz, M. D., *Associate Professor of Parasitology, Associate in Medicine, The Jefferson Medical College of Philadelphia; Special Consultant, U. S. Public Health Service, Communicable Disease Center, Atlanta, Ga.* 296 pages; illustrated. The Blakiston Co., Philadelphia, Pa., publishers, 1950. Price $4.25.

The author states: "This manual is intended as a lecture and laboratory guide for students in their course in medical parasitology. It does not, and is not intended to, replace textbooks which are essential for parasitologists. Medi-

cal schools, however, do not train parasitologists." To achieve this end, much of the material is presented in abbreviated or outline form or in telegraphic style. The book is of real value in that the tables and charts include much recent information not generally available in texts, including data on drugs, insect repellents, and insecticides. Dosages are tersely specified in each case. As a rule the charts and tables are excellent, but many of the illustrations are poor, one or two of the figures being unrecognizable. The keys are of too limited a scope to be of much use in the identification of parasites, but are of value in enabling students to learn some of the criteria used in classification and identification. The clinical descriptions are at times sketchy. The toxicity of certain anthelmintics are not adequately stressed. This book will be useful if supplemented by the use of standard texts, laboratory guides, and adequate lecture material. It has real merit as a review of the subject of parasitology, particularly insofar as it enables one quickly to find notes on the most recent advances in the therapy of parasitologic infections and in the control of arthropods of medical importance.—*Maj. R. Traub, MSC, U. S. A.*

Hematology for Students and Practitioners, by Willis M. Fowler, M. D., *Professor of Internal Medicine, University of Iowa, Iowa City, Ia.*, with a chapter by Elmer L. DeGowin, M. D., *Associate Professor of Internal Medicine, University of Iowa, Iowa City, Ia.* 2d edition, revised. 535 pages, illustrated. Paul B. Hoeber, Inc., New York, N. Y., publishers, 1949. Price $8.50.

This second edition of a valuable textbook on hematology presents the usual aspects of hematologic disorders, physiology of hematopoiesis and blood dyscrasias, in a thoroughly accurate, simple, and satisfactory manner. The sections on hematologic methods, the transfusion of whole blood and blood derivatives, and leukemia are particularly good. The author states in his preface "that hematology is a part of internal medicine, rather than a specialty in itself, and the subject has been presented in this light, stressing the clinical and the therapeutic aspects of the various diseases." This is not a textbook for research hematologists. The concept of "hyperheparinemia" and the effect of radiation on the hematopoietic tissues are completely omitted although the latter is of considerable importance in current times when atomic warfare is conceivable. The illustrations are in general excellent; the bibliography is limited but adequate. The book is highly recommended for the medical student and for the practitioner of medicine.—*Commander E. P. Cronkite, MC, U. S. N.*

Selected Studies on Arteriosclerosis, by Rudolf Altschul, M. U. Dr., *Professor of Histology, University of Saskatchewan, Saskatoon, Canada.* 182 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1950. Price $5.50.

In discussing the morphologic changes in experimental arteriosclerosis and in the correlation of his own investigations with others engaged in parallel work, the author offers an engaging series of adducements to one willing to persevere through the obtuseness of his writing. To the reviewer the almost exclusively morphologic approach to the problem seems anachronistic. With broader vistas opening continually in hormonal and enzymatic concepts of the origin of disease, the days of the morphologists appear to be numbered. This work is best categorized by this sentence taken from the preface: "It makes no claim of being of assistance to the clinician or even to the hospital pathologist."

—*Lt. R. K. Moxon, MC, U. S. N.*

Endodontia, by Bernhard Gottlieb, M. D. (University of Vienna), D. M. D. Hon. (University of Bonn), LL. D. Hon. (Loyola University, Chicago); *Professor of Oral Pathology and Dental Research, Baylor University College of Dentistry, Dallas, Tex.; Honorary Member of the American Association of Endodontists; formerly Professor and Head of Department of Histologic Research, College of Dentistry, University of Vienna;* Seth Lee Barron, D.D.S., *Assistant Professor of Root Canal Therapy, Baylor University College of Dentistry, Dallas, Tex.;* and J. Hobson Crook, D. D. S., *Associate in Dental Research, Baylor University College of Dentistry, Dallas, Tex.* 177 pages; illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1950. Price $6.

This book is mainly a report of the histologic findings resulting from experimental root canal treatment in dogs. Numerous short articles on subjects related to endodontia are included. The book is not composed of chapters, but is divided only by topic headings. It is well illustrated, and 63 of the figures are exceptionally good photomicrographs. The authors believe that their most important contribution is the histologic demonstration that cementum will bridge over the outlet of pulp canals when dentine powder is used in root canal therapy. Some of the theories presented in certain sections are contrary to generally accepted competent opinion, but on the whole the book is a stimulating addition to dental literature. Because this is not a complete textbook on endodontia, but rather an informal discussion of certain phases of the subject, it is recommended only as supplemental reading for those having particular interest in this field.—*Commander R. A. Colby, DC, U. S. N.*

The Esophagus and Pharynx in Action, A Study of Structure in Relation to Function, by William Lerche, M. D., *Fellow, American College of Surgeons; Founder Member and Honorary Member of the American Association for Thoracic Surgery, University of Minnesota, Minneapolis, Minn.* 222 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1950. Price $5.50.

The primary purpose of the author is a discussion of the closing mechanism of the cardia of the stomach. The book also includes a description of the physiologic activity and function of the esophagus in the process of swallowing and regurgitation. This is an excellent monograph on the function of the esophagus and is of interest to those concerned with gastroenterology and surgery of the esophagus.—*Commander T. C. Ryan, MC, U. S. N.*

Principles of Public Health Administration, by John J. Hanlon, M. S., M. D., M. P. H., *Associate Professor of Public Health Practice, School of Public Health, University of Michigan, and Chief Medical Officer and Associate Chief of Party, Bolivia, the Institute of Inter-American Affairs.* 506 pages; illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1950. Price $6.

No one should expect to read through this comprehensive book in 1 evening or 20 evenings. The principles are those taught by the nation's leading schools of public health and may be safely quoted as references. Each chapter encompasses a large section of civilian health department work and is particularly complete in the background of laws and customs which have accompanied our social advances in the United States. The use of graphs and charts with explanations of the strength and weakness of each helps in understanding the complexities of modern State and city governments within our country. Two chapters are worthy of special comments. The chapter entitled "Personnel Factors in

Public Health" contains many practical suggestions which would improve the human relationships of health departments and also in other local governmental departments; and the one entitled "Public Relations in the Public Health Program" points the way for selling better service and reaching more persons and explains why some worthy programs have failed in the past. The material in part 3 presents both sides of the perplexing relationships involving public health and private enterprise. In this controversy, the more understanding health officials read both sides, the more surely a workable solution can be found. The fair and impartial discussions will be of much help to administrators in health departments and to those working toward this goal.

—*Col. G. Orth, MC, U. S. A.*

The Principles and Practices of Rehabilitation, by Henry H. Kessler, M. D., Ph. D., in collaboration with other authors. 448 pages; with 132 illustrations and a colored plate. Lea & Febiger, Philadelphia, Pa., publishers, 1950. Price $9.

Treatment of the patient as a whole rather than of a disease or injury is the central theme of this book. Dr. Kessler has chosen his 20 collaborators well. Together they have put out a well-written volume, general in scope and content, which should serve as a useful guide. Orthopedic and plastic surgeons and workers in the various fields of physical therapy, prosthetics, and social welfare will find this volume useful in providing a background and an insight into the problems of their coworkers. The text is not limited to the rehabilitation of a single type of patient, but through the use of a number of contributors, each a specialist in his own field, runs the gamut from the chronically ill to the deformed. The text is divided into two parts entitled (1) "Principles" and (2) "Practice." The scope of the book is so broad that there is little or no room for detailed instructions. It achieves its aim and presents an integrated picture of the armamentarium available to those medical and lay persons who deal extensively with the physically and mentally handicapped.

—*Lt. Comdr. C. F. Orofino, MC, U. S. N.*

Recent Advances in Ocular Prosthesis, by J. H. Prince, F. B. O. A., F. S. M. C., F. R. M. S., F. Z. S. A companion volume to "Ocular Prosthesis." 155 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1950. Price $4.

This small text brings one up to date on plastic artificial eyes, augmenting the author's previous book "Ocular Prosthesis." Six of the ten chapters deal with different methods of making conjunctival impressions; properties of materials used; preparation of artificial irides; and the fabrication and processing of acrylic eyes. A smaller, but adequate, portion of the text deals with the newer meshed and peg implants outlining the different operative procedures and the advantages of the various types. The procedures are well illustrated. An outline of treatment for some unusual cases of contracted fornices and sockets is presented. A few pages are devoted to plastic surgery and the consideration of permanently embedded prostheses. Detachable facial and orbital prostheses are discussed in general terms. The book is well written and adequately indexed. Although it is not the practice in this country for opthalmologists to concern themselves directly with the making of artificial eyes, nevertheless they could find in this small text much about the subject that they should know, and also information that would enable them to express an intelligent opinion about the finished fitted prosthesis.

—*Commander P. J. Giotta, MC, U. S. N.*

Surgery of the Eye: Injuries, by Alston Callahan, B. A., M. S. (Ophth.), M. D., F. A. C. S., *Professor of Ophthalmology, Medical College of Alabama, and Director, Thigpen-Cater Eye Hospital, Birmingham, Ala.; formerly Chief, Eye Section, Northington General Hospital, Tuscaloosa, Ala.* 240 pages; 367 illustrations. Charles C Thomas, Publisher, Springfield, Ill., 1950. Price $11.

This text is of special importance to the military ophthalmologist. The author served in an Army ophthalmic center during World War II where he was able to study 3,000 men with injuries of the eye and adnexa. Since that time he has had further opportunity to study eye injuries and surgical procedures at the Thigpen-Cater Eye Hospital, Birmingham, Ala. The surgical procedures described are those which gave the best results in his hands. The operative sketches and eye photographs are excellent. The timing of this book is fortunate now that our military hospitals are receiving many eye casualties from Korea. This text is highly recommended to all military ophthalmologists. They will profit from the various technics described in this book and pass these benefits on to their patients.—*R. Adm. C. A. Swanson, MC, U. S. N.*

Essentials of Ophthalmology, by Roland I. Pritikin, MC., F. A. C. S., F. I. C. S., *Eye Surgeon, Rockford Memorial, Winnebago County and Swedish-American Hospitals; Consulting Ophthalmologist, St. Anthony Hospital, Rockford, Ill.* 561 pages; 215 illustrations including 18 subjects in colors. The J. B. Lippincott Co., Philadelphia, Pa., publishers, 1950. Price $7.50.

The author has maintained rigid faithfulness to his title. This fidelity has resulted in what is almost an encyclopedia of ophthalmic terms. This book provokes the question, "Who, precisely, are the beneficiaries of such condensations?" The pace is too staccato for teaching undergraduate medical students, and the more serious student of ophthalmology demands more than synopsized brevity. The general practitioners with whom I have discussed this problem are not interested in even brief treatments of such matters as optics, orthoptics, and spectacle fitting. They are interested in ocular first aid, differential diagnosis of acute ophthalmic pathology, and the ocular manifestations of organic and neurologic disease. These subjects could have been treated more fully in this volume. The section on physical optics uses three dimensional drawings to refreshing advantage. The illustrations throughout are excellent.

—*Commander E. A. Hynes, MC, U. S. N.*

Practical Gynecology, by Walter J. Reich, M. D., F. A. C. S., F. I. C. S., *Attending Gynecologist, Cook County Hospital; Professor of Gynecology, Cook County Graduate School of Medicine; Attending Gynecologist, Fantus Clinics, of the Cook County Hospital; Assistant Professor of Gynecology, Chicago Medical School; Attending Gynecologist and Obstetrician, Grant Hospital; Attending Gynecologist, Fox River Tuberculosis Sanitorium; Consulting Gynecologist, Hazelcrest General Hospital;* and Mitchell J. Nechtow, M. D., *Associate Attending Gynecologist, Cook County Hospital and the Fantus Gynecologic Clinic; Assistant Clinical Professor of Gynecology, Cook County Graduate School; Associate in Gynecology and Obstetrics, Chicago Medical School; Attending Gynecologist and Obstetrician, Norwegian-American Hospital.* 449 pages with 187 illustrations, including 55 subjects in color. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1950. Price $10.

The general practitioner, resident, and intern frequently inquire concerning the proper approach to the management and treatment of office-type procedures in gynecology. This clear and concise volume on practical gynecology should

implement and extend the sketchy and hurried instruction noted in some medical school curricula and hospital teaching programs. The excellence and clarity of composition of the many fine drawings, colored plates, and other reproduced materials enhance the well-written easily read text. This book is based on a popular series of conferences and demonstrations given by the authors in their postgraduate course in gynecology at Cook County Hospital, Chicago, Ill. It offers a simple guide to the technic of office-type practice founded on years of gynecologic out-patient clinical work. Controversial and esoteric material which so often confuse the novice and general practitioner, have been omitted for the most part, making for greater clarity, easier reading, and assimilation. It should serve as a firm foundation and incentive to the study of more detailed and comprehensive textbooks on the subject.

In addition to elaborating the technic of office gynecology, including systematic routine and thorough examination, laboratory tests, biopsies, cytology, diagnosis, and management of common disorders, great stress is placed on the fact that treatment and instruments recommended are those commonly available or easily improvised in the physician's office or clinic thus often obviating costly equipment and hospitalization. Of special value are the chapters on the practical approach to gynecologic diagnosis, the early detection of cancer, common gynecologic complaints, premarital examinations and consultations. The absence of a bibliography detracts from the value of the book but the index is especially complete.—*Col. E. A. Zimmermann, MC, U. S. A.*

Medical Physics, Volume II. Editor-in-Chief, Otto Glasser, Ph. D., F. A. C. R., *Diplomate in Radiological Physics, American Board of Radiology; Professor of Biophysics, Frank E. Bunts Educational Institute; Head, Department of Biophysics, Cleveland Clinic Foundation; Consultant, U. S. Veterans' Administration, Washington, D. C.*, and editorial assistant Jessie C. Tucker, *Cleveland, Ohio*, and associate editors. 1,227 pages; 978 illustrations. The Year Book Publishers, Inc., Chicago, Ill., publishers, 1950. Price $25.

Volume II contains almost 1,200 pages of encyclopedic information on applications of modern physical science to medical and biologic phenomena. Many of the 181 contributors are recognized authorities in the specialties on which they write. The book is expensive but well printed and highly recommended for ready reference to the numerous and intricate specialties of modern medical biophysics. Extensive references are appended to each chapter for those inclined toward serious investigation. Prospective purchasers should note, however, that Volume II complements and supplements the first volume. Chapters and subjects treated in Volume I appear in Volume II only by title; but where rapid and important advances have been made since 1944, the new material only appears in Volume II with reference to the introductory chapter of Volume I. The books will be of greatest value when purchased and used together.

Particular emphasis has been placed on atomic radiation phenomena and especially on the biologic and medical applications. Several chapters of introductory information on particle accelerators, radiation counters, isotopes, and so forth, have been included. Roentgenology, radiation, optics, and photography have been extensively treated. The variety of other subjects is such that many difficult editorial problems were undoubtedly encountered in the allotment of space, and many readers may feel that their own special interests have been too lightly dismissed. Colloid and surface phenomena, for instance, were allotted 2 pages of historical generality; whereas 6 pages and about 20 illustrations were devoted to types of hypodermic needles and how to sharpen them. Impartial reflection, however, should lead the reader to the conclusion that such individual

prejudices must necessarily be discounted in a book of this scope; and that, in the final analysis, the editors and authors have produced a comprehensive and masterful introduction to a very large number of specialties in biophysics.

—Lt. Col. M. E. Freeman, MSC, U. S. A.

The Management of Obstetric Difficulties, by Paul Titus, M. D., *Obstetrician and Gynecologist to the St. Margaret Memorial Hospital, Pittsburgh; Consulting Obstetrician and Gynecologist to the Shadyside Hospital, Pittsburgh; Secretary of the American Board of Obstetrics and Gynecology; Member Reserve Consultants Advisory Board, Bureau of Medicine and Surgery, United States Navy (Captain, MC, U. S. N. R.).* 4th edition. 1,046 pages; with 446 illustration and 9 color plates. The C. V. Mosby Co., St. Louis, Mo., publishers, 1950. Price $14.

This work has always been unique in that it states its purpose in the title and then proceeds to show the how, why, and when with the least unnecessary verbiage. The problems currently debated in the recent literature are adequately treated and the author's position stated. The entire subject is completely covered and yet there is no effort to make the volume encyclopedic. This edition has 50 more pages than the third edition, as a result of sandwiching new paragraphs into the old text. It is hoped that the author can see fit completely to rewrite the next edition or it is likely to become so voluminous as to defeat its purpose. This is one of the few books addressed to physicians in active practice rather than to medical students. As such, it is a valuable addition to the personal library of anyone concerned with the welfare of the pregnant and parturient woman. Based as it is on the clinical work of the author and his associates, it has a personal flavor, too often missing in modern texts and reference books.

—Commander R. E. Crowder, MC, U. S. N.

Principles of Orthodontics, by J. A. Salzmann, D. D. S., F. A. P. H. A., *Associate Attending Dentist and Head of Orthodontics at the Mt. Sinai Hospital, New York; formerly head of the Dental Service at the New York City Vocational Schools; Associate Editor of the American Journal of Orthodontics; Editor of the New York Journal of Dentistry.* 2d edition. 887 pages; 533 illustrations. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1950. Price $15.

This book represents a landmark in the progress of dental literature. The use of a vast amount of source material combined with personal experiences gained at the chair has lead to a balanced text. There are numerous illustrations.

The basis of orthodontic science is not merely a matter of making or designing a mechanical appliance which will push or pull a tooth or groups of teeth into certain positions but rather a system for understanding and removing causes which lead to the various oral and facial deformities. Other means than mechanotherapy should be tried reserving the use of mechanical appliances as a last resort. For these reasons the author devoted the major portions of his book to development and growth of the head, developmental anatomy and physiology of the face and the jaws, development of the dentition, the endocrine glands in relation to dentofacial deformity, treatment of incipient malocclusion, and preventive orthodontics and public health. Only in a small section near the end of this work does the author allow us to view with him some of the more popular forms of appliances in mechanotherapy, but even here, there is great attention to detail. A clear picture of the practical application of the labial and lingual aid applianees, the Angle edgewise appliance, the Tweed method, the Johnson twin wire appliance, and the Norwegian system for treatment of malocclusions is given.

The author warns those who practice orthodontics to beware of too rapid tooth movement because of the attendant destruction of tooth vitality and osseous structures. The book is of value to those who do not practice orthodontics because it helps to evaluate and understand the significance of causative factors in malocclusion and because it stresses early detection and timely interception of incipient developmental anomalies and thereby enables us to direct the patient to the physician or dentist for proper therapy. To the orthodontist in active practice this book should serve as a guide for diagnosing and treating the pathologic states that present themselves to him. The general use of this text in dental colleges is to be recommended but an expert teacher of orthodontics must elaborate on the text to clarify certain parts which may prove too complex for the dental student. For the graduate dentist studying the specialty of orthodontics this book will be indispensable. A large list of references is included.

—Capt. M. Dicher, U. S. A. F. (DC).

An Atlas of Human Anatomy, by Barry J. Anson, Ph. D., *Professor of Anatomy, Northwestern University Medical School.* 518 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publishers, 1950. Price $11.50.

This valuable new anatomical atlas should prove most welcome to medical students and clinicians alike. The preparation of an anatomy text is always a sizeable undertaking and Dr. Anson and his colleagues have expended much time and effort compiling and editing this work which "represents the fruition of a long term project."

The atlas should serve equally well as a dissecting manual or as a surgical anatomy reference book.

The illustrations for the most part are refreshingly new and original. Those few drawings which are modifications of plates taken from other standard works are clearly identified as such. A minimum of diagrammatic illustrations are used. The labeling of the various structures in the plates is in large, easily read, hand lettering. There is none of the crowding present which is so often encountered in some of the older texts. The explanations given at the bottom of the pages containing illustrations are clear and concise. There are relatively few colored plates—probably because of the expense involved in reproducing them. Their lack, however, does not detract from the general value of the book.

A splendid series of drawings of the internal and middle ear structures are outstanding features of the atlas. The inclusion of the many illustrations showing anatomic variables is a valuable innovation. Variations in the types of origin of the branches and tributaries of important arteries, veins, and nerves are well illustrated. Variations in the form and structure of the thoracic duct, vermiform appendix, stomach, pelvic colon, and hepatic pedicle are also clearly portrayed. The drawings dealing with hernias of various types are especially well done and should prove of definite value to surgeons. Dr. Anson has included many figures taken from his articles published in various scientific journals.

—Col. E. L. Kehoe, MC, U. S. A.

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